The Dissertation Committee for Hyun Gu Kang Certifies that this is the approved version of the following dissertation:

Kinematic and Motor Variability and Stability during Gait: Effects of Age, Walking Speed and Segment Height

Committee:

Jonathan B. Dingwell, Supervisor

Lawrence D. Abraham

Joseph P. Cusumano

Lisa Griffin

Waneen W. Spirduso

Kinematic and Motor Variability and Stability during Gait: Effects of Age, Walking Speed and Segment Height

by

Hyun Gu Kang, B.S.; M.S.

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Dedication

in memory of Kevin P. Granata

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Kinematic and Motor Variability and Stability during Gait:
Effects of Age, Walking Speed and Segment Height

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Hyun Gu Kang, Ph.D.
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Supervisor: Jonathan B. Dingwell

To understand how falls occur during walking in older adults, we need to understand how the nervous system maintains stability, and how aging affects walking. Four studies were conducted to better understand the effect of age on gait.

Older adults display higher gait variability compared to young adults, possibly because of their slower walking. We compared gait stability at multiple controlled walking speeds. Greater gait variability in healthy elderly existed independent of slower walking. Their diminished strength and flexibility partly explained this difference.

To explain slower walking in the elderly, some have suggested that muscle weakness and stiffness may force people to walk slower. Others have suggested that people choose to walk slower to be more stable. We compared dynamic stability of gait at multiple speeds. Healthy older adults also exhibited more stability at slower speeds, yet walked at speeds comparable to young adults despite the lower strength and flexibility. Therefore, weakness and stiffness may not force healthy older adults to walk slower.

The goal of the nervous system during walking may be to maintain stability of superior segments. We tested whether superior segments are more stable than inferior segments during walking. Superior segments exhibited less orbital stability during preferred walking speed, in contrast to previous suggestions. This highlighted the importance of trunk control during gait.

The effects of aging on the fluctuations in the muscle activity during gait are not well understood. We quantified the stride-to-stride fluctuations of EMG as a measure of muscle activation patterns in state-space. Variability increased with speed except in the gastrocnemius. Orbital stability was less in older adults, suggesting that deviations in the EMG amplitude pattern were not readily corrected. Less local stability was seen in older adults, suggesting that older adults were more sensitive to perturbations.

Together, these findings suggest that trunk control is important during gait. Strength and flexibility deficits help explain higher variability and lower stability in older adults. Future work will need to address the effect of strength interventions, neurophysiological decline on gait stability and fall risk.

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Chapter 1: Introduction

Falls are the primary cause of accidental death and injury among adults over age 65 (Alexander, Shepard et al. 1992; Murphy 2000). Injuries from falls are debilitating, and billions of dollars are spent every year in related health care (AHRQ 2005). Most falls in older adults occur during daily activities such as walking (Blake, Morgan et al. 1988; Tinetti, Doucette et al. 1995; Niino, Tsuzuku et al. 2000). In order to understand falls in older adults that occur during walking, it is important to understand the strategies used by the nervous system to maintain stability, and how aging affects walking.

The major age-related changes related to walking can be categorized into the following groups: physical changes such as muscle weakness, reduced range of motion, and the decline of sensory function (visual, tactile, vestibular); and behavioral changes such as slower walking speed, increased double-support phase, increased variability (Winter, Patla et al. 1990; Bassey, Fiatarone et al. 1992; Dobbs, Lubel et al. 1992; Maki 1997). Some have suggested that the changes in musculoskeletal function may cause people to walk with these behavioral changes (Larish, Martin et al. 1988; Elble, Thomas et al. 1991). Others have suggested that these behavioral changes reflect the adaptation to these physical changes, and people may *choose* these changes (Murray, Kory et al. 1969; Winter, Patla et al. 1990; Alexander 1996). Despite this discussion, the causes of these behavioral changes are not clear. The difficulty lies in being able to identify these underlying causes.

Among the behavior changes with aging, gait variability has received much attention because increased variability has been shown to be related to aging and fall risk (Lord, Lloyd et al. 1996; Maki 1997; Hausdorff, Rios et al. 2001). Older adults display higher gait variability compared to healthy young adults (Owings and Grabiner 2004), but the cause of this higher variability is unclear. Older adults typically walk slower (Alexander 1996), but the gait of healthy young adults also becomes more variable as they walk at slower speeds (Winter 1983; Dingwell and Marin 2006). This suggests that increased gait variability observed in older adults may be a result of slower walking speed. However, other factors associated with aging, such as loss of strength and flexibility may also cause increased gait variability. Clarifying this issue involves separating the effects of walking speed on gait variability from that of age and other factors. To test whether higher variability in older adults can be attributed to walking speed alone, gait variability in both young and older adults must be quantified at multiple controlled walking speeds. By quantifying gait variability at different speeds for both young and older adults, we can determine if increased variability seen with older adults is caused merely from slower walking speed, or if other factors are contributing to the increased variability in older adults.

Slower walking speed is associated with various clinical conditions in the elderly (Alexander 1996). To explain slower walking speed commonly observed in older adults, some have suggested that age-related physical factors such as muscle weakness and stiffness may force people to walk slower (Larish, Martin et al. 1988; Elble, Thomas et al. 1991). This is supported by the evidence that slower walking speeds are correlated with previous falls in older adults (Alexander 1996). Others have suggested that people *choose* to slow down to allow for more double-support and stability (Murray, Kory et al. 1969; Winter, Patla et al. 1990). This is supported by the evidence that slower walking is more stable in young adults (Dingwell and Marin 2006). This issue can be clarified by quantifying how stability varies with walking speed and also with strength and flexibility in older adults.

The control of the upper body is important, as the trunk segment consists of over half of body mass (Winter 1990), and greatly influences the dynamics of the rest of the body. Not simply a mass to be transported by the legs, the upper body performs a multitude of functions related to support and stabilization during walking (Winter, MacKinnon et al. 1993; Prince, Winter et al. 1994; Kavanagh, Morrison et al. 2006). Research suggests that control of superior segments takes precedence over other segments of the body during walking. Shocks from walking are absorbed as they move up the kinematic chain (inferior to superior segments) toward the head (Ratcliffe and Holt 1997). This suggests that the stability of superior segments is prioritized over inferior segments. Also, the relationship between the stability of superior and that of inferior segments may be affected by aging, as body composition, shape and mass ratio change. This can be tested by comparing the stability of body segments relative to their "segment height" in the body in young and older adults.

Beside sensory function, motor function is also affected by aging. Higher force variability found in older adults may indicate increased motor errors (Tracy and Enoka 2002), and may result in increased variability in motor patterns. Current methods to describe the effects of aging on the motor system during gait are usually limited to that of behavior of individual muscles during walking, and do not adequately describe the interaction of multiple muscles, or the interactions of motor patterns over multiple strides. Because of its multivariate and cyclic nature, motor patterns are well suited to be described using state-space methods. In particular, the robustness of walking motor patterns to recover from fluctuations can be assessed by quantifying the stability of motor outputs measured using electromyography, to describe age-related changes in motor patterns.

In this dissertation, we have explored these issues in the context of treadmill walking. The background and previous works related to these issues are described in Chapter 2. Healthy young and older adults were recruited to walk on a treadmill while their motion and muscle activity were measured. Four sets of analyses were completed, as described in Chapter 3. In the first study (Chapter 4) we investigated the effect of speed on gait variability in young and older adults. In the second study (Chapter 5) we investigated the effect of waking speed on gait stability in young and older adults. In the third study (Chapter 6) we examined the effect of the segment height on stability during walking. In the fourth (Chapter 7) we explored the robustness of motor patterns. Summary and conclusions can be found in Chapter 8.

SPECIFIC AIMS AND HYPOTHESES

Specific Aim 1: to determine whether the increased gait variability in older adults is caused by slower walking, by quantifying kinematic variability as a function of walking speed and age group

Hypothesis 1: Older adults exhibit greater variability across different walking speeds on these dependent measures than young adults:

a) Stride time
b) Step length
c) Step Width
d) Hip flexion/extension angle
e) Hip abduction/adduction angle
f) Hip internal/external rotation angle
g) Knee flexion/extension angle
h) Knee varus/valgus angle
i) Knee internal/external rotation angle
j) Ankle dorsiflexion/plantarflexion angle
k) Ankle pronation/supination angle
l) Ankle internal/external rotation angle
m) Trunk tilt angle
n) Trunk obliquity angle
o) Trunk rotation angle
p) Trunk COM velocity in anterior-posterior (AP) direction

q) Trunk COM velocity in medial-lateral (ML) direction
r) Trunk COM velocity in vertical (VT) direction

Specific Aim 2: to determine why healthy older adults choose to walk slower, by quantifying local and orbital stability of the trunk as a function of walking speed on a treadmill in healthy young and older adults

Hypothesis 2: (A) Local and (B) orbital stability of the trunk segment is less at faster walking speeds in young adults.
Hypothesis 3: (A) Local and (B) orbital stability of the trunk segment is less at faster walking speeds in older adults.
Hypothesis 4: (A) Local and (B) orbital stability of the trunk segment is less at all walking speeds in older adults compared to young adults.

Specific Aim 3: to determine whether the stability of superior segments is greater than in inferior segments, by quantifying local and orbital stability as a function of segment height in healthy young and older adults at their preferred walking speed

Hypothesis 5: (A) Local and (B) orbital stability is less in inferior segments than superior segments in both young and older adults.
Hypothesis 6: (A) Local and (B) orbital stability is less in all segments in older adults compared to young adults.

Specific Aim 4: to determine the effect of walking speed and age on the stability of EMG linear envelopes

Hypotheses 7: Older adults exhibit greater variability across different walking speeds on these dependent measures than young adults:

a) *Vastus lateralis* EMG linear envelope
b) Hamstrings EMG linear envelope
c) *Gastrocnemius* EMG linear envelope
d) *Tibialis anterior* EMG linear envelope

Hypothesis 8: (A) Local and (B) orbital stability of EMG linear envelopes is less at faster walking speeds in young adults.
Hypothesis 9: (A) Local and (B) orbital stability of EMG linear envelopes is less at faster walking speeds in older adults.
Hypothesis 10: (A) Local and (B) orbital stability of EMG linear envelopes is less at all walking speeds in older adults compared to young adults.

To fulfill these aims, walking motion and EMG from the left leg were measured during walking in young and older adults. Isometric leg strength and passive joint range of motion were also measured.

SIGNIFICANCE

To prevent falls in the elderly, it is important to understand the aging process. Through better understanding of both healthy aging and complications associated with disease processes, we may be able to define and diagnose more effectively such deteriorations and apply the appropriate interventions. To that end, the results of this study have further elucidated the causes underlying the behavioral changes associated with aging. Fulfilling Aim 1 made this the first study to examine gait variability comprehensively at multiple controlled speeds in both young and older adults. The answers have clarified the reasons underlying an increase of gait variability in older adults. Fulfilling Aim 2 has helped to distinguish whether slower walking may be a conscious choice in older adults. The insights gained from fulfilling Aim 3 have helped us understand how control of body motion is prioritized. In completing Aim 4, we have increased our understanding of how aging affects the coordination of muscle activities during walking. The findings will help develop better understanding of the aging process, and aid in developing clinical interventions to prevent falls in the elderly.

DELIMITATIONS

Only healthy young and older adults who reported no orthopedic or neurological conditions and have no history of falls were included in this study. The findings on these healthy elderly may not extend to those with a history of falls or other pathologies. Also, in this study, the response of a walking person only to small perturbations was investigated. The results from this study cannot necessarily be inferred to determine the ability to recover from large perturbations during walking, or the basin of attraction for global stability. The subjects were asked to walk only at speeds with which they were comfortable, and experienced only the small perturbations present during normal walking that come from sources such as irregularities on the treadmill, neural noise, motor errors, etc. This study was not designed to test the limits of stability and the performance of the subjects. It also does not consider the effects of vision, vestibular function, or cognitive processing speed.

As a cross-sectional design, rather than a longitudinal design, this study did not directly control for subject history in the older adult group. Any differences found may not necessarily be attributed only to the aging process.

LIMITATIONS

Because treadmill walking enforces a constant walking speed, the use of a treadmill may artificially reduce the natural variability (Wank, Frick et al. 1998), and enhance the local dynamic stability (Dingwell, Cusumano et al. 2001) of walking kinematics. However, in theory treadmill and overground walking are mechanically identical as long as the belt speed is assumed constant (van Ingen Schenau 1980). Although many belt-type treadmills exhibit intra-stride speed variations (Savelberg, Vortenbosch et al. 1998), subjects in this study were tested on a Woodway treadmill, which incorporates a direct-drive servomotor to minimize this problem. Even though every attempt was made to minimize possible mechanical differences, subtle psychophysical differences may exist between the current treadmill walking and natural overground walking (van Ingen Schenau 1980). These differences such as the lack of optic flow present in overground walking, but missing during the treadmill walking trials, and the size of the treadmill that can constrain motion, may have influenced the results.

Subject selection was not accomplished in a purely random manner, and young subjects were recruited to height- and weight-match the older adults. Recruitment of subjects was accomplished using flyers and advertising, to recruit subjects as "randomly" as possible.

Limb and joint motions were measured using surface markers. Because soft tissue moves differently with respect to bones, measured motion also reflects the motion of the soft tissue as well as bones. Because the only way to avoid this issue is to surgically attach motion markers to the bone, which is painful and costly, we instead used multiple redundant markers on each segment to help minimize the effects of soft tissue movement. Soft tissue movement might be minimized using a low body-mass index population, but this may not be representative.

Manual isometric strength testing used in this study may not fully quantify leg strength. Forces were measured while asking the subjects to perform maximum voluntary contractions. Isometric measurement does not account for the power-generation capacity. These measurements are subject to motivation and other factors. Although interpolated twitches may provide a more accurate maximum force capability of a muscle, motor neurons for the large muscles of the leg cannot be accessed superficially.

DEFINITION OF TERMS

Stability

The definition of stability in the context of walking dynamics and fall prediction is rather contested, and many different definitions exist depending on the context and use. Unsteadiness or variability is often used to refer to the lack of stability as clinical predictors of falls. More discussion can be found in section 5 of the following literature review in Chapter 3. Here, we use a definition of stability based on engineering dynamics, as a system's response to perturbations, especially its capacity to deal with perturbations. Stability can be quantified using the response of the system to perturbations in terms of the behavior of its state variables, the variables that describe its behavior (Full, Kubow et al. 2002). Here, we focus on local stability. Local stability refers to the behavior of a system in response to very small perturbations. It describes whether the system will return toward its current state or not, after receiving a small perturbation. This is in contrast to global stability, which defines whether a system can receive a perturbation of any size and still recover.

Local stability can be defined in different ways, which will be discussed in section 5 of the literature review in detail. Briefly, local dynamic stability is quantified by estimating how a system would respond to a small perturbation, by comparing the current trajectory of a system to a nearby trajectory. It describes if the system on a particular trajectory was perturbed onto a nearby neighboring trajectory, whether the system would converge back toward the original trajectory, or diverge away. For periodic systems, a special case can be defined, known as orbital stability, which refers to the tendency of the system to return toward the system's natural cycle called the "limit cycle," after a perturbation. Measures of orbital stability, known as Floquet multipliers, can distinguish young adults, healthy older adults, and recurrent fallers (Granata and Lockhart 2006).

Chapter 2: Literature Review

This section provides the background information on falls in the elderly, and describes the previous studies that led to the four studies described in Chapter 1. The literature review consists of six parts, covering the following topics:

1. Falls in the elderly
2. Walking Speed
3. Variability in Walking
4. Head stability
5. Measuring Stability
6. Walking and the Nervous System

1. FALLS IN THE ELDERLY

1.1 Epidemiology

Falls are a significant and extremely costly health problem for older adults (Englander, Hodson et al. 1996; Fuller 2000). Current epidemiology reports that more than one-third of adults over 65 fall each year (Hornbrook, Stevens et al. 1994; Hausdorff, Nelson et al. 2001) in the community and over 60% in nursing homes (Fuller 2000). These falls are a primary cause of death and injury among this population (Alexander, Shepard et al. 1992; Murphy 2000).

1.2 Costs

Hip fractures are the one of the most common injuries associated with falls (Fuller 2000). Health care costs associated with hip fractures in elderly cost over $7 billion health care dollars in 2003 (AHRQ 2005). The percentage of the United States population over age 64 is expected to increase to over 20% or 86.7 million by year 2050 (Bureau 2004). Given current population models and rise in health care costs, fall-related injuries may cost over $25 billion by year 2050 (Marin 2004).

1.3 The importance of studying gait

Most falls in older adults occur during some form of locomotion (Blake, Morgan et al. 1988; Tinetti, Doucette et al. 1995; Niino, Tsuzuku et al. 2000). Reduced function in the balance control system and in skeletal muscle strength are observed with age (Grimby and Saltin 1983; Fiatarone and Evans 1993). Some have argued that these are the reasons that gait patterns are modified (Hahn, Lee et al. 2005). Older adults, 63% of those institutionalized in hospitals or long-term care facilities, or 8-19% in the community, have difficulty ambulating, making gait performance a useful clinical assessment tool of function (Alexander 1996).

Aging results in changes in motor behavior and decreased walking stability. To prevent the negative impact of the falls in the elderly, such as the loss of quality of life as well as financial burdens, it is important to understand how falls occur during walking and how they can be prevented. To that end, we need to study how stability is maintained during walking, and what the underlying mechanical and physiological mechanisms are.

1.4 Age-related changes in gait

Age related changes in relation to walking include physical factors such as weakness and reduced range of motion; behavioral factors such as slower walking speed, increased double-support phase, increased variability; sensory factors such as degradation of visual, tactile, and vestibular function (Winter, Patla et al. 1990; Bassey, Fiatarone et al. 1992; Dobbs, Lubel et al. 1992; Maki 1997). These observations are closely linked to each other, and to fall risk, but a big distinction needs to be made between physical factors from behavioral factors to understand how they are related to falls. Physical factors such as range of motion, strength and vestibular function are factors that can physically limit a person, and in some sense, are fixed such that people cannot change them readily. Their effects are easier to understand. Muscle weakness would prevent a person from generating enough force to counteract a fall-causing perturbation. Likewise, reduced range of motion would also prevent a person from responding to perturbations.

Some intervention studies that include strength training and stretching have been shown to reduce fall risk (Barnett, Smith et al. 2003; Liu-Ambrose, Khan et al. 2004), but others have not. Exercise programs have not reduced fall risk in women (Ballard, McFarland et al. 2004) or fall risk score in women (Liu-Ambrose, Khan et al. 2004). This may be because strength training only seems to affect "pre-frail" or healthy older adults (Faber, Bosscher et al. 2006).

In contrast, behavioral factors such as slower walking speeds and increased variability are produced by the motor system, and their relationship with fall risk is not as clear, even though they may be correlated to fall risk. These observations may be a result of the physical capabilities, or that of people strategically improving their walking stability. The physical factors such as weakness and low range of motion may be causing slower walking speed and increased double-support, or these behavioral factors may be proactive measures used by people to increase their stability. Stated differently, these behavioral changes may be indications of gait issues that develop with age, and the inability to walk normally, putting them at risk of falls. Or they may be compensatory, active strategies to maintain stability to deal with some destabilizing factor.

Despite much discussion on what causes falls, these issues have not been resolved in the literature. To clarify the reason for falls in the elderly, we need to control for the effects of these physical factors and behavioral factors better.

2. VARIABILITY IN WALKING

2.1 Variability and Fall risk

Of all the gait variables, variability has been used extensively to study falls in the elderly. Variability is typically quantified as standard deviations (SD) or coefficients of variation (CV) of particular measures (Winter 1983; Öberg, Karsznia et al. 1993; Öberg, Karsznia et al. 1994). In young healthy adults, stability and variability vary differently with speed, where stability decreases with speed, while variability increases as people walk slower or faster than their preferred speed (Dingwell and Marin 2006). Some argue that a reduction in the ability to control motion leads to less consistency and increased variability of that motion. Applied to walking, this might translate into an increased likelihood of falling. Some studies found that higher variability, especially step width variability, predicted an increased risk of falls (Lord, Lloyd et al. 1996; Maki 1997; Hausdorff, Rios et al. 2001), although one study found that trunk acceleration variability, and not step width variability, could predict falls (Moe-Nilssen and Helbostad 2005).

However, even though increased variability seems to be related to decline of motor control as used in clinical settings, some kinematic variables may not need to be tightly controlled. Scholz and Latash et al. have shown that while some variables are tightly controlled, manifested in the form of low variability, others are not, leading to higher variability (Scholz, Schöner et al. 2000; Latash, Scholz et al. 2001). This was termed their "uncontrolled manifold hypothesis." Therefore, slower walking seems to lead to an increase in variability (Winter 1983; Öberg, Karsznia et al. 1993; Öberg, Karsznia et al. 1994), but increases or decreases in variability may depend on which variables are being examined and the context (Marin 2004), and not all of them may be related to fall risk.

2.2 Relationships to Walking Speed and Age

In healthy adults, the kinematics of walking are more variable as people walk slower or faster than their preferred speed (Winter 1983; Öberg, Karsznia et al. 1993; Öberg, Karsznia et al. 1994; Dingwell and Marin 2006; Jordan, Challis et al. 2006). Many studies that investigated the relationship of gait speed and variability were conducted using self-selected paces of "slow," "normal," "fast" walking speeds that were measured from overground walking (Winter 1983; Öberg, Karsznia et al. 1993; Öberg, Karsznia et al. 1994; Moe-Nilssen and Helbostad 2005). When fixed walking speeds are imposed using a treadmill, speed and variability in young adults display a quadratic "U-shape" relationship (Yamasaki, Sasaki et al. 1984; Yamasaki, Sasaki et al. 1991; Dingwell and Marin 2006; Jordan, Challis et al. 2006). Healthy older adults exhibit increased step width variability compared to young adults at their preferred walking speed (Owings and Grabiner 2004; Owings and Grabiner 2004). This is especially noticeable when older adults walk over irregular surfaces (Menz, Lord et al. 2003; Menz, Lord et al. 2003). Older adults also tend to walk slower, and slower walking is more variable in young adults, which suggests that the increased variability comes from the slower walking speed.

2.3 Summary and Next Steps

To summarize, gait variability increases with age and slower walking speeds. However, it is not clear if the increase in gait variability in older adults is simply due to them walking slower, or can be attributed to other age-related factors. It has been suggested that older adults walk slower to improve their local dynamic stability even though it may increase their walking variability (Dingwell and Marin 2006). This reasoning assumes that older adults exhibit a similar relationship between variability and walking speed as young adults, but this assumption must be tested. This assumption states that (A) young and older adults have the same relationship between variability vs. speed, and older adults walk at a slower speed, leading to increased variability (Figure 2-1). However, other possibilities can explain the increased variability in older adults. For example: (B) Young and older adults have a similar variability vs. speed relationship, but older adults are more variable at all speeds. The increased variability seen in older adults may arise from increased variability at all walking speeds. (C) Variability in older adults is more sensitive to speed, than in young adults. Older adults become more variable as they deviate from their preferred speed compared to young adults. (D) Young and older adults have a different variability vs. speed relationship, where the variability vs. speed curve are offset in speed from each other. Clarifying which of these possibilities (as well as other possibilities) may be correct would involve separating the effects of walking speed on gait variability from that of age. To test whether higher variability in older adults can be attributed to walking speed alone, gait variability in both young and older adults needs to be quantified at multiple controlled walking speeds. By quantifying gait variability at different speeds for both young and older adults, we can determine if increased variability seen with older adults is caused merely from slower walking speed, or if other factors are contributing to the increased variability in older adults.

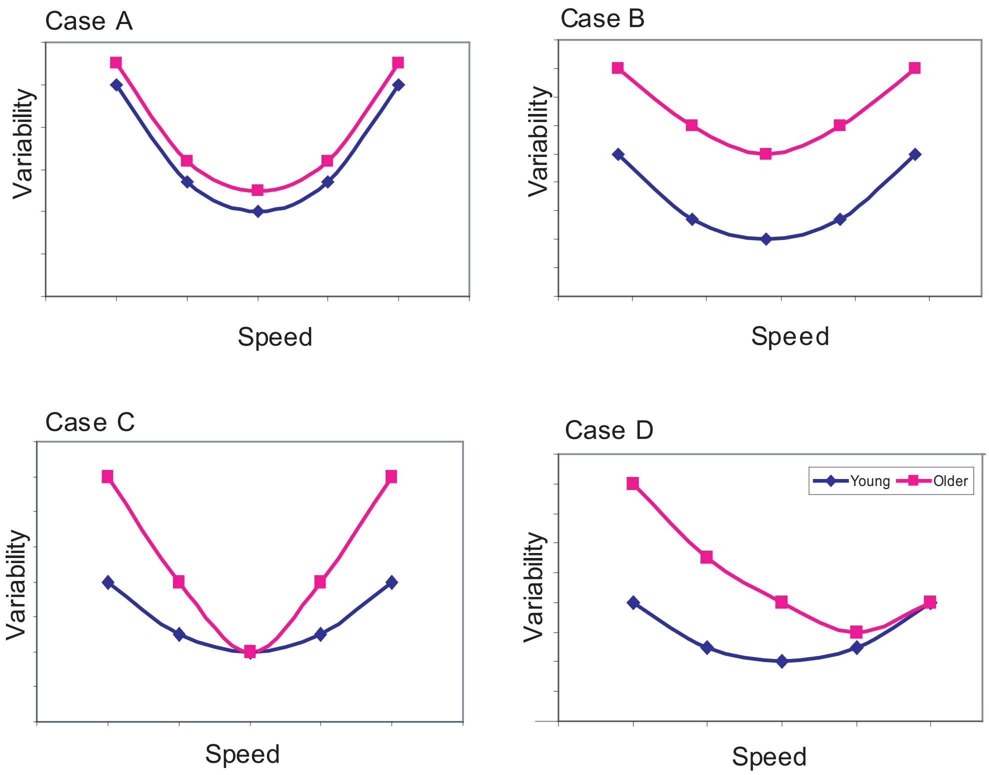


Figure 2-1. Various Possible Relationships between Variability and Speed

3. Walking Speed

3.1 Observation of slower walking speed

Decreased walking speed is associated with many different musculoskeletal, neurological and cardio-respiratory conditions and diseases, such as Alzheimer's disease, vascular dementia and depression (Alexander 1996). Slower walking is observed along with slower cadence and a shorter stride length in hemiparetic stroke patients (Titianova and Tarkka 1995), Huntington's disease patients (Thaut, Miltner et al. 1999), hip replacements due to bone tumors (Kawai, Backus et al. 2000), diabetic neuropathy (Dingwell and Cusumano 2000), history of falls (Kerrigan, Lee et al. 2000), and peripheral vascular condition (Gardner, Forrester et al. 2001). As common as this phenomenon of slower walking speed may be, it is not clear why older adults walk slower.

With advanced age, slower walking speed is associated with low daily activity level, sense of gait unsteadiness and previous falls (Alexander 1996). Preferred walking speed decreases even in otherwise healthy older adults by 0.2% to 1.6% per year in this group (Himann, Cunningham et al. 1988; Sato and Ishizu 1990; Dobbs, Charlett et al. 1993). Some have noted that the changes are marked by shorter strides rather than a change in cadence (Alexander 1996). Controlling for walking speed and subject height, the spatio-temporal differences in young and older adults are very small or do not exist (Alexander 1996). Others have found shorter strides in older adults than young adults even when velocities were artificially matched (DeVita and Hortobagyi 2000).

Slower walking speed may exist because of physical limitations such as metabolic, cardiovascular, or nervous deficits associated with the conditions described above. This may also be caused by behavioral adaptations to these conditions. And yet, it is difficult to attribute any specific causes to the changes and differences in walking speed with aging, especially in those who are displaying "higher - level gait disorder," displaying no specific medical conditions that may affect gait (Herman, Giladi et al. 2005).

3.2 Slower walking speed and fall risk

Slower walking speed may be related to various clinical conditions, it is not clear if it is related to fall risk. Some have found that gait speed was not predictive of prospective fall risk (Feltner, MacRae et al. 1994; Maki 1997; Hausdorff, Rios et al. 2001). Yet others have found that slow walking speed does predict a higher risk of falls (Luukinen, Koski et al. 1995; Bergland, Jarnlo et al. 2003). Nevertheless, none of these studies provide a causal relationship between walking speed and the risk of falls. Rather, walking speed seems to be a marker of an underlying pathology in older adults (Marin 2004). For example, success in rehabilitation after hospital admission can be predicted by faster walking speed (Alexander 1996). Healthy older adults walk faster than those less healthy, but increasing walking speed via strength training has not been shown to decrease fall risk (Chandler, Duncan et al. 1998). Also, faster walking may only increase trips and falls (Pavol, Owings et al. 1999; Pavol, Owings et al. 2001). Thus, although slower walking speed is indicative of decline in function, its value as a fall predictor is yet unclear.

3.3 Physical Factors and Slower Speed

Several studies attempted to find some evidence that musculoskeletal and metabolic changes *cause* older adults or patient groups to walk slower. The energetic cost of walking increases with age (Mian et al. 2006; Kuo et al. 2007). This may be due to the increased cost of maintaining stability in the elderly.

Gait speed of stroke patients could be explained by the range of joint moments the subjects produced during walking, but this explanation did not account for subjects' actual strength or power generation capabilities (Olney, Griffin et al. 1994). It may be that the lower joint moments are simply due to walking slower. Controlling for gait speed, young and older adults displayed differences in joint torques and powers (DeVita and Hortobagyi 2000), but these measures of gait kinetics do not necessarily reflect an age-related decrease in strength. Knee extensor torque generation capability in stroke patients explained over 50% of the variance in gait speed (Bohannon 2001). Yet in older men with leg weakness, hip extensor torque explained only 37% of gait speed variance (Burnfield, Josephson et al. 2000). These studies are limited in the scope of variables measured, and only describe correlations between strength and walking speed. Intervention studies improve on these studies. Strength training increased walking speed in older adults (Chandler, Duncan et al. 1998; Holviala, Sallinen et al. 2006). However, the improved speed is still less compared to a younger population. Thus, physical changes associated with age, especially strength, may be a factor in slower walking speeds in older adults. However, the observed differences associated with age do not fully explain the slower walking speeds in older adults.

3.4 Fear of Falls and Slower Speed

Slower walking speed can not only be attributed to physical constraints, but also to feeling “safer” or more “steady” or “stable” (Winter, Patla et al. 1990; Gardner and Montgomery 2001; Menz, Lord et al. 2003). Maki et al showed that fear of falls is related to gait speed in older adults (Maki 1997). This suggests that slowing down is a precautionary measure, perhaps because older adults may feel unsteady and thus uncomfortable at normal speeds of young adults. Similar observation can be made with young adults, as healthy young adults also walk more slowly when walking on a slippery surface or with eyes closed as a precaution. Dingwell and Marin demonstrated that local dynamic stability does increase with slower walking speeds in young adults, suggesting that slowing down can improve stability during walking (Dingwell and Marin 2006). However, this finding has not been demonstrated in older adults, or other clinical populations at risk of falls.

3.5 Summary and Next Steps

To summarize, slower walking has been observed in a variety of clinical conditions and with age. Some have suggested that this slower walking speed stems from a physical constraint that prevents them from walking faster. Although these factors such as strength help explain slower walking speed, they have not been shown to be the causative factors of slower walking, and do not fully explain slower walking speed in older adults. Others have suggested that slower walking may be a pro-active strategy to improve stability. Slower walking is more stable in young adults, but this has not been demonstrated in older adults.

To understand better the reasons for slower walking in older adults, we need to separate the limiting effects of physical deterioration from the effects of any adaption to fear of falls or a sense of stability. This better understanding of the aging process may help us ameliorate the disease process. However, the interaction of these physical and behavioral changes is not well known. This can be by testing if the slower walking speed is in fact more stable in older adults as well, in relation to their strength. Slower walking in older adults also may be more stable, suggesting that older adults choose to walk slower. If slower walking speed is less stable, it may suggest that older adults are forced to walk slower with increased risk of falls.

4. Trunk Stability and Importance of Superior Segments

The trunk segment consists of over half of body mass (Winter 1990), and greatly influences the dynamics of the rest of the body. Not simply a mass to be transported by the legs, the trunk musculature performs functions related to support and stabilization during walking (Winter, MacKinnon et al. 1993; Prince, Winter et al. 1994; Kavanagh, Morrison et al. 2006). Active control of the trunk motion is believed to enable stability to be maintained during walking (Winter, MacKinnon et al. 1993; Prince, Winter et al. 1994). Because of its mass, if the movement of the trunk segment is not controlled well, then the rest of the body may follow, perhaps leading to a fall.

4.1 Sensory Systems in the Head

Clinical observations note that the head movement is always tightly controlled during various tasks, such as standing, dynamic movements (Pozzo, Levik et al. 1995), and walking (Holt, Ratcliffe et al. 1999; Cromwell, Schurter et al. 2004). Research suggests that control of superior segments takes precedence over other segments of the body during walking. Shocks from walking are absorbed as they move up the kinematic chain (inferior to superior segments) toward the head (Ratcliffe and Holt 1997), keeping superior segments stable. By the time they reach the head, accelerations dampen to ~20% of that measured at the shank level (Kavanagh, Morrison et al. 2006).

These observations suggest that one goal of the nervous system is to prioritize the stabilization of the superior segments. This may be because visual and vestibular sense organs are located in the head. It has been hypothesized that lack of head control may indicate risk of falls (Holt, Ratcliffe et al. 1999). Control of head movement comes from compensating for trunk movements as well as movements of more inferior segments (Cromwell, Newton et al. 2002).

4.2 Summary and Next Steps

Given the evidence that shocks from walking are absorbed as they move up the kinematic chain (inferior to superior segments) toward the head, it follows that after the head segment, the trunk would be second most controlled, and pelvis the next, and so on.

This then suggests that the stability of body segments would also follow the order of segment height, where superior segments are more stable than inferior segments. However, not much is known about the control of multiple segments with respect to each other, or how age affects these relationships. Only a few studies examined the effect of age on trunk movement. There is some evidence that older men walk with less trunk acceleration than young adults after adjusting for walking speed (Kavanagh, Barrett et al. 2004). In the only study that explored the stability relationship of multiple segments, Floquet multipliers (FM) based on the vector from the heel to the center of mass at each step were found to be larger in older adults with a history of falls compared to healthy older adults, and FM was even less in young adults (Granata and Lockhart 2006). However, it is not clear how aging or other clinical conditions affect the control of the trunk segment relative to lower segments.

5. WALKING AND THE NERVOUS SYSTEM

5.1 Role of neuromuscular system in Gait stability

Because most falls occur during walking in the elderly, it is important to study how the neuromuscular system responds to perturbations that may induce falls. Falls occur after a perturbation because the necessary corrective responses to counteract the effects of the perturbation are not provided. Normally, the neuromuscular system senses the perturbation, and activates the motor pathways, which in turn activates the muscles in order to produce corrective action. If any of these functions are impaired, through sensory loss (such as vestibulopathy, peripheral neuropathy), cognitive impairments (Alzheimers, Huntington's), motor impairments (Parkinson's or multiple sclerosis), or at the muscle level (such as sarcopenia and decreased flexibility), a person may not be able to correct for the effect of the perturbation and a fall may occur. A person must generate both the correct motor patterns to facilitate stable walking, and also the corrective responses to perturbations.

Humans naturally receive small perturbations during walking. These perturbations can be from external mechanical sources such as the unevenness of the floor or a slippery floor, as well as from internal motor sources, such as force fluctuations from the muscle. In order to understand how these continuous perturbations are handled by the nervous system, their effect on fluctuations of the nervous output need to be studied, both in terms of statistical properties, and their response to perturbations over time.

5.2 Perturbations and Challenging Walking

Perturbation experiments have been used to elucidate the response of the nervous system during walking. In these experiments, sudden perturbations are given to a subject while walking, or "challenging" walking situations are created, such as walking on a narrow beam. These studies have elucidated how the nervous system responds to experimental manipulations of the nervous system during walking.

In a series of experiments, sudden deceleration perturbations were applied when a person was walking on a treadmill (Berger, Dietz et al. 1984; Dietz, Quintern et al. 1985; Dietz, Quintern et al. 1985; Dietz, Quintern et al. 1986; Dietz, Quintern et al. 1987). By measuring Hoffmann reflex (H-reflex) during walking, Dietz et al described reflex modulation during different parts of the gait cycle in young adults. Early ipsilateral response with 65-75ms latency seems to be responsible for repositioning the foot, to correct for the unexpected positioning of the foot due to the perturbation. Early contralateral and late ipsilateral responses were found to compensate for body displacements. Based on the latencies of the reflex actions, they concluded that these responses initiate from group II and III afferents rather than group I afferents. This was based on demonstrating that ischemic nerve blockage of group I afferents did not affect these reflex actions (Berger, Dietz et al. 1984), or their effects on cerebral potentials (Dietz, Quintern et al. 1985).

H-reflexes during walking were reduced during mid-stance in older adults compared to young controls (Chalmers and Knutzen 2000). In both the older and the young adult groups, the H-reflex amplitude was negligible at the time of heel contact, rose to a maximum shortly after mid-stance, decreased rapidly as toe-off neared, and then was negligible during swing. During mid-stance of walking, the older adults had a smaller H-reflex size, compared to young adults despite no significant difference in H-reflex size between the two age groups while standing. The smaller H-reflex size during the stance phase of walking may reflect changes in central reflex mechanisms that may suppress stretch reflex contribution to ankle extensor neural drive and ankle stiffness in elderly persons during walking (Chalmers and Knutzen 2000), or decline in monosynaptic reflex function.

This result suggests that the blocking of group I afferents to the cerebral cortex found by Dietz et al. may be more pronounced with aging. The smaller H-reflex during stance phase in older adults suggests that any corrective short-latency reflexes may be suppressed, and therefore any monosynaptic corrections to perturbations are also suppressed. However, the effect of age has not been studied on polysynaptic corrective mechanisms described by Berger, Dietz and Quintern studies, while Chalmers and Knutzen do not discuss the interaction of multiple muscles between young and older adults. One of the difficulties in this type of method is that motor neurons for most leg muscles cannot be accessed using a surface stimulating electrode. Also, although these studies reveal how the nervous system deals with mechanical perturbations, they do not discuss how response behavior fluctuates over multiple strides.

In an experiment comparing walking on a narrow beam compared to normal ground, the level of muscle activation throughout the gait cycle was increased in the distal musculature, namely *gastrocnemius* and *tibialis anterior*, among older adults, but not in young adults (Brown, Gage et al. 2002). The authors suggested that there is a unique strategy of distal control for the regulation of gait in older adults. This was in contrast to the prevailing theory (Winter and Yack 1987; Tang, Woollacott et al. 1998), where the proximal muscles are the main stabilizers during walking where balance challenges were anticipated, or "proactive" as opposed to "reactive" tasks to unexpected challenges that involve distal muscles. These studies suggest that more distal muscles are favored with less reliance on sensory feedback during walking in older adults.

However, it is not clear if this possible lesser reliance on sensory feedback is due to the decline of monosynaptic reflex function (Chalmers and Knutzen 2000) or a change in strategy due to some other factor. Also, although Brown et al described the coordination of the different muscles in young and older adults, they did not describe how the muscle activity fluctuates between strides, or how these subjects respond to perturbations during their "proactive" state.

5.3 Variability of EMG Patterns

During locomotion, EMG amplitudes can vary considerably, compared to kinematics, which do not vary as much (Winter and Yack 1987; Trank, Chen et al. 1996; Grasso, Bianchi et al. 1998; Grasso, Zago et al. 2000; Ivanenko, Grasso et al. 2002). Some have suggested that the motor system prioritizes the control of limb kinematics to adapt to the environment, and regardless of how it is accomplished by the muscle activation patterns (Shen and Poppele 1995; Borghese, Bianchi et al. 1996; Grasso, Bianchi et al. 1998). Because of the redundancy of the muscular system, the various muscle activation patterns that produce similar kinematics will activate different muscles at different times in different ways, more variability is expected, as well as observed, in the EMG linear envelopes compared to kinematics. Another possibility is that the complex motor patterns are low-pass filtered by the mechanical system, smoothing out the variations.

The loss of motor units with age contributes to increased force variability due to motor unit firing synchronization. This has been observed in small muscles in isometric conditions, such as the first dorsal interosseus muscle (Delwaide 1986; Vaillancourt, Larsson et al. 2003). This effect is likely to be more pronounced in the large muscles involved in gait, variability of EMG linear envelopes as well as increasing kinematics variability.

While kinematic variability is influenced by speed, it is not clear how variability of EMG amplitude as a measure of muscle activation varies with speed or age. Coefficient of variation (CV) of EMG signals in *vastus lateralis* and hamstrings muscles decreased with speed, while in *gastrocnemius* and *tibialis anterior* CV stayed the same with speed (Shiavi, Bugle et al. 1987). However, CV's reported do not give us any information on the changes in EMG amplitude during different walking speeds. Burst duration and onset latency became more variable with slower walking, in older adults, but amplitudes were not reported (Chung and Giuliani 1997). In healthy young adult men, no relationship was found between the amount of motor unit synchronization in various leg muscles and the speed of walking (Hansen, Hansen et al. 2001). However, this finding may not extend to older adults, where any increase in the amount of synchronization may influence the observed variability of EMG amplitudes.

In summary, the current evidence suggests that slower walking is associated with both increased variability in amplitude and timing of EMG patterns, and only one study described the increased CV of amplitude. However, existing studies have used self-selected speeds of “slow,” “fast,” etc., or imposed fixed walking speeds, without accounting for differences in body size, or an individual’s preferred walking speed. This can be addressed by imposing fixed controlled walking speeds in relation to each subject’s preferred walking speed. This would give us a more systematic look into the fluctuation of motor patterns during gait in young and older adults. In Aim 4 of this study, we addressed this by comparing the variability of EMG signals in both young and older adults across multiple controlled walking speeds using a treadmill.

Using statistical moments to describe motor variability during walking is common in literature, but these methods ignore how individual fluctuations in motor outputs relate to each other over time. In most of the studies, EMG signals are ensemble-averaged over multiple strides, ignoring the time-course of how EMG varies over many consecutive strides. Many investigators also do not study the coordinated activity in a group of muscles. Often, signals from many muscles are studied only one at a time, or the few studies that use multivariate methods only provide a qualitative view (Craik and Oatis 1995). However, because walking is cyclic, it lends itself to be studied using analyses that account for its cyclic nature, which is only beginning to be explored.

5.4 Cyclic Muscle Activation

The isolated spinal cord can create a coordinated rhythmic movement in the absence of supraspinal input via a putative group of spinal neurons referred to as a central pattern generator (CPG). This phenomenon has been demonstrated extensively in spinal and decerebrate cats (Shik and Orlovsky 1976), as well as other vertebrates (Grillner

1975). The presence of a CPG in humans has not been demonstrated very strongly, but many clinicians are trying to use it in rehabilitation of spinal cord injury patients (MacKay-Lyons 2002). If walking motor patterns are generated by a central pattern generator, can the muscle activation patterns be explained as just a few individual patterns? Using principal component analysis, EMG activation profiles of up to 32 muscles in the leg and trunk have been explained by as few as 5 principal components (Patla 1985; Davis and Vaughan 1993; Olree and Vaughan 1995; Ivanenko, Poppele et al. 2004). The 5 principal components accounted for 60-90% of the variance in the EMG data during walking (Ivanenko, Poppele et al. 2004). Cats exhibit 5 synergies of muscle activation patterns that account for >91% of the muscle activity during standing in response to horizontal translations in many different directions (Ting and Macpherson 2005) as quantified using nonnegative matrix factorization (Lee and Seung 1999). These studies suggest that a limited number of motor patterns combine to form both transient and cyclic behavior, possibly indicative of few independent pattern generators.

5.5 State-space description of the Nervous output

A small number of signals can reconstruct most of the EMG signals of multiple muscles during walking. And, because of their rhythmic, cyclic nature, the motor output from the spinal cord during walking may be described as a stable cycle, or as a limit cycle. A system exhibits a stable “limit cycle” behavior if it displays a cyclic or rhythmic behavior that can recover from a perturbation and go back to the cyclic or rhythmic behavior (see section 6 for more details).

The literature use of principal component analysis to reduce multi-muscle EMG signals in human walking suggested that 5 principal components are enough to describe all of the muscle activation patterns at different walking speeds (Ivanenko, Poppele et al. 2004). This is supplemented by the Global false nearest-neighbors studies, where 5 state variables are sufficient for describing the entirety of human walking kinematics (Dingwell and Cusumano 2000). These studies suggest that 5 state variables may be sufficient to describe and capture limit-cycle-like behavior of the muscle activation patterns during walking, but this has not been demonstrated in the literature. A state-space description of EMG state space has been used to describe walking, but its use has been limited to a description of the difference between level and uphill walking (Jansen, Miller et al. 2003). Here, the differences were quantified using a statistical clustering method.

The limited number of muscle activation patterns, described in state space, allows us to track cycle-to-cycle fluctuations of these activation patterns. Fluctuations in EMG linear envelope have not been described over multiple strides. By tracking multiple muscles at once, state-space descriptions may be able to track changes due to aging, fatigue, or therapeutic intervention better than by studying the fluctuations in one muscle at a time.

5.6 Next Steps

Common in the literature, using variability to describe the characteristics of motor pattern during walking tells us the statistical property of the fluctuations overall, but these methods ignore the how individual fluctuations in motor outputs relate to each other over time, or from one stride to the next. In most studies, EMG signals are ensemble-averaged over multiple strides, ignoring the time-course of how EMG varies over many consecutive strides. These methods do not adequately describe the interaction of multiple muscles, or the fluctuations of motor patterns over time during walking. Often, signals from many muscles are studied only one at a time, or the few studies that use multivariate methods only provide a qualitative view (Craik and Oatis 1995). Only few studies described the coordinated activity in a group of muscles. Methods employing principal components analysis to describe the activity of multiple muscles at once (Ivanenko, Poppele et al. 2004; Ivanenko, Poppele et al. 2006) do not account for the stride-to-stride fluctuations in EMG. Same issue is seen for a multivariate clustering method to describe walking uphill vs. on level ground (Jansen, Miller et al. 2003). Cycling of synergistic muscle activity has been demonstrated during low-level sustained contractions over time, but these studies use isometric contractions (Kouzaki, Shinohara et al. 2002; Kouzaki and Shinohara 2006).

To address these limitations of previous methods, we can use state-space description of EMG signals to describe walking, that accounts for their multivariate and temporal behavior. Unfortunately, the use of state-space methods to describe the use has been limited to a description of the difference between level and uphill walking (Jansen, Miller et al. 2003). Because of its multivariate, somewhat periodic nature, motor patterns during gait are well suited to be described using state-space methods. In particular, the robustness of motor patterns during walking can be assessed, by quantifying the stability of motor outputs measured using electromyography to describe age-related changes in motor patterns. A state-space description allows tracking of cycle-to-cycle fluctuations of these activation patterns in multiple muscles over multiple strides. By tracking multiple muscles at once, state-space descriptions may be able to track changes due to aging, fatigue, or therapeutic intervention better than by studying the fluctuations in one muscle at a time. Stability of muscle activation patterns provides a new way of quantifying the fluctuations in motor outputs. These methods allow us to quantify if burst patterns during one stride are related to those in subsequent strides, and if they reflect inner workings of the nervous system differently from kinematics. In addition to the study of kinematic variability vs. speed and age in Aims 1, a study of variability of EMG signals during gait as a function of speed and age is also indicated.

6. MEASURING STABILITY

Because falls occur mostly during walking, we must study how people maintain stability during walking. Statistical measures such as variability are often used to predict falls, but they can only tell us correlations, rather than causes. They do not quantify how the locomotor system responds to perturbations that may cause a stumble or a fall (Dingwell and Cusumano 2000) and thus, do not give us a mechanistic view into how stability is maintained during walking. More appropriate measures that quantify actual walking *stability* are needed before we can study the underlying mechanical and neural mechanisms that maintain stability during walking.

6.1 Assessing stability

In clinical settings, heuristic measures such as variability and standing stability are often used. Heuristic measures are derived from clinical experiments and from correlations of fall risk to various measures. Many different measures have been used to predict the incidence of falls as a way to predict whether a specific patient is at risk for fall-related injuries (Hornbrook, Stevens et al. 1994; Tinetti, Doucette et al. 1995; Hausdorff, Rios et al. 2001). Performance in functional tests such as the Berg balance test have been successful in predicting falls (Mechling 1986; Duncan, Weiner et al. 1990; Duncan, Studenski et al. 1992; Shumway-Cook, Baldwin et al. 1997; Dennis 1999; Nordt, Sachatello et al. 1999; Lin, Hwang et al. 2004). Static posturography (Topper, Maki et al. 1993; King, Judge et al. 1994; Prieto, Myklebust et al. 1996; van Wegen, van

Emmerik et al. 2002; Laughton, Slavin et al. 2003; Lafond, Corriveau et al. 2004) is also often used.

Because gait variables describe gait function, temporal-distance measures of gait have been used to evaluate gait function and dysfunctions in the elderly and fall prediction (Gabel and Nayak 1984; Heitmann, Gossman et al. 1989; Ferrandez, Pailhous et al. 1990; Elble, Thomas et al. 1991; Leiper and Craik 1991; Yack and Berger 1993; Judge, Roy B. Davis et al. 1996; Maki 1997; Brach, Berthold et al. 2001; Hausdorff, Nelson et al. 2001; Menz, Lord et al. 2003; Owings and Grabiner 2004). Spatio-temporal measures give an overall picture of walking performance, but inter-subject variability is large in these measures. This may prevent accurate prediction of fall risk for a specific individual (Hahn and Chou 2005).

Perhaps because these measures are relatively easy to obtain from patients, they have been developed into indices used to predict fall risk. However, they in themselves do not tell us *why* certain older adults are at risk of falls, and what could prevent falls. To understand falls better, we need to study how fall-causing perturbations are handled by the neuromuscular system.

6.2 Variability measures

Variability measures are also widely used in fall predictions. Step width variability during walking has been shown to be a very useful predictor of fall risk (Maki 1997; Hausdorff, Rios et al. 2001; Owings and Grabiner 2004; Owings and Grabiner 2004). These variability measures predict falls, and they may be related to the ability to maintain steady gait, but what do they tell us about how people maintain stability? Often it is assumed that steadiness (or the lack of variability) is a sign of stability, and larger variability indicates instability. Thus large variability during walking is considered unstable walking.

However, is variability necessarily a negative factor in gait? Increased variability may mean that a person cannot maintain a steady gait because the person is not focused on walking, or is executing incorrect motor commands during walking, thus putting him or her at risk for falls. However, increased variability may indicate that a person is adapting to the environment, and changing his or her motor patterns to negotiate the terrain and other perturbations. Variability that indicates the inability to perform the locomotor task is of concern, but variability can also indicate the adaptability of a person to deal with the environment. Variability may also reflect the capacity to execute a wide range of equally successful step behaviors without falling. Conversely, little or no variability may mean that a person's walking is very robust despite the influence of the environment, or that a person's capacity to adapt to the environment is limited and he or she is thus more likely to fall.

Using variability as a measure, the two cannot be distinguished. This may be why too little or too much step width variability is correlated with falls in both cases (Brach, Berlin et al. 2005). Variability is a statistical measure that describes the spread about the mean, and as such, provides no theoretical basis for equating statistical spread about the mean to stability or how people maintain stability during walking. Even though correlations are found between variability and risk of falls, this in itself does not provide a causative mechanism of falls, or explain how stability is maintained during walking.

6.3 Stability in State Space

In order to assess how people maintain walking stability, we need a way to measure dynamic stability and quantify the person's ability to adapt and respond to the perturbations from the environment. The body receives external perturbations as slips, bumps, etc. as we walk. We can measure how the locomotor system responds to these perturbations.

We track whether the effects of a perturbation grow or shrink over time in the system. If the effects of a perturbation become smaller by its effect being absorbed and corrected, this indicates stability, or the person's ability to keep walking. If the effects of perturbation grow and are not corrected, it indicates instability, and may lead to a fall. To track the effects of perturbations on the body, we need to track the variables that describe the behavior of the system, or "state variables," such as position, velocity, rotations, etc. If not all of the state variables are available, they can be substituted by time-delayed copies of variables that are available (a technique known as delay-embedding), and still preserve the basic dynamics of the system (Takens 1981). Because perturbations can affect different state variables differently, all at once, in subtle ways, we need to consider all state variables at the same time, in what is called state space. This is akin to using phase plots in clinical settings, where a phase plot of knee and ankle angles reveal differences between healthy and pathologic populations although each of them by themselves did not (McClay and Manal 1997). To fully track the effects of perturbations, we need to track them in state space.

To track the effects of perturbations, we need to first know how the system behaves without perturbations. Only then can we track deviations of the state variables from this unperturbed state. In walking, where each gait cycle is similar to each other, and normally follows a specific pattern, we can average the behavior of the system over multiple strides and consider this mean gait cycle as our "unperturbed" gait cycle. It seems that the person is trying to stay on this average path over many strides, and that the system is trying to return to this path or trajectory after a perturbation. This trajectory is called a "limit cycle" or an "attractor." A person's walking described as a trajectory in state space can be compared to the limit cycle, and its stability can be determined by whether the walking trajectory is moving toward or away from the limit cycle.

Stability can be understood in two distinct ideas: local stability and global stability. Local stability refers to the behavior of a system in response to very small perturbations. It describes whether the system will return toward its original state or path or not, after receiving a small perturbation. Global stability describes how large of a perturbation a system can receive and still recover. This is too difficult to quantify in humans *in vivo*, because it requires providing extreme range of perturbations to people, which can be dangerous (Pavol, Owings et al. 2001; Smeesters, Hayes et al. 2001). In this study, we will only consider local stability. Local stability can be defined in different ways, which will be discussed in below.

6.4 State-space Construction and Reconstruction

As described above, state-space is a set of state variables, variables that describe the dynamics or "states" of a system. The perturbation dynamics of standing and walking can be estimated by examining the trajectories of the system's movements in state space. When properly defined, the state variables of a system fully describe its behavior. For example, a mechanical system consisting of a single point mass with 1 degree of freedom can be described by a 2nd order differential equation:

$$F(t) = k\mathbf{x} + b\dot{\mathbf{x}} + m\ddot{\mathbf{x}} \qquad (2\text{-}1)$$

where $\mathbf{x}$, $\dot{\mathbf{x}}$, and $\ddot{\mathbf{x}}$ are position, velocity and acceleration, k, b, and m are system parameters (i.e. stiffness, damping, and mass), and $F(t)$ is the external force applied to the system. This equation constrains the acceleration to be a function of position and velocity. Therefore, the system's position and velocity can be used to define the two state variables that are sufficient to fully describe the dynamics of the system, $S(t) = [\,\mathbf{x}, \dot{\mathbf{x}}\,]$ (Pai and Patton 1997). An extended 3-dimensional rigid body has three translational plus three rotational degrees of freedom. The dynamics of this 2nd order system can therefore be fully described in terms of these 6 degrees of freedom [$x\ y\ z\ \theta\ \phi\ \psi$] and their time derivatives $[\dot{x}\ \dot{y}\ \dot{z}\ \dot{\theta}\ \dot{\phi}\ \dot{\psi}]$, thus constructing a 12-dimensional state space:

$$S(t) = [x, y, z, \dot{x}, \dot{y}, \dot{z}, \theta, \phi, \psi, \dot{\theta}, \dot{\phi}, \dot{\psi}] \qquad (2\text{-}2)$$

However, sometimes some state variables cannot be measured. As demonstrated numerically by Packard (Packard, Crutchfield et al. 1980) and proven by Takens (Takens 1981), information contained in unobserved state variables can be attained by using time-delayed copies, or time-derivatives of existing state variables. A state-space formed from these other state variables is said to be "re"constructed,

$$S'(t) = [x(t), x(t+\tau), x(t+2\tau), \cdots x(t+(d_E - 1)\tau] \qquad (2\text{-}3)$$

where τ is the time delay, and d_E is the embedding dimension. Appropriate time-delays can be calculated using the first zero-crossing of the autocorrelation function, or the first minimum of average mutual information function (Dingwell and Cusumano 2000). Embedding dimensions can be calculated using various methods, such as correlation dimension (Grassberger and Procaccia 1983) or global false nearest-neighbors algorithms (Kennel 1992). Reconstructed state spaces are "dynamically equivalent" to the original state space of the system, preserving information on the inherent dynamics of the system.

6.5 Mean Divergence and Lyapunov Exponent Methods

One previously developed approach for quantifying the "*local stability*" of walking kinematics (Dingwell and Cusumano 2000) is based on the calculation of local divergence exponents (Rosenstein, Collins et al. 1993; Kantz and Schreiber 1997), which quantify how a system's states respond to very small (i.e., "local") perturbations continuously *in real time*. The perturbation affects different state variables differently, where their responses can be written as exponential functions, described using a set of

Lyapunov exponents (λ) for each state variable that specify which are stable or unstable. Positive λ indicates that a perturbation will keep growing over time, or instability, while negative λ indicates that a perturbation will become smaller over time, or stability. The largest λ in a system, or λ_{max} is used as a metric of stability, because a system is only as stable as its least stable state variable. This is analogous to a chain that is as strong as its weakest link.

Lyapunov exponent methods were further developed to calculate sensitivity to initial conditions in aperiodic[1] systems and time-series data (Fig 2-2). In the context of data analysis, λ_{max} , maximum Lyapunov exponent, is estimated from local divergence properties that are averaged over the entire data set, and will give an overall indication of sensitivity to small perturbations, or local stability, in a system. Previous studies demonstrated that patients with diabetic neuropathy improve their local stability *because* they slow down (Dingwell and Cusumano 2000). Also slower walking speeds lead to improved local stability, while faster speeds lead to greater instability (Dingwell and Marin 2006; England and Granata 2006), but measures of gait variability do not predict local stability during walking (Dingwell and Marin 2006).

Euclidean distances between neighboring trajectories in state space are computed as a function of time and averaged over all original pairs of initially nearest neighbors. For each point $S(t)$ on the state-space trajectory, the nearest neighboring point $S(t^*)$ on an adjacent trajectory (excluding points on the same trajectory) was determined, forming the j^{th} pair of nearest neighbors. Euclidean distances between each pair of subsequent points on the two trajectories were then calculated (Figure 2-4C). For this j^{th} nearest neighbor pair of $S(t)$ and $S(t^*)$, this formed a vector of Euclidean distances $d_j(i)$:

$$d_j(i) = \left\| S(t + i\Delta t) - S(t^* + i\Delta t) \right\|_2 \qquad (2\text{-}3)$$

[1] Aperiodic: not periodic; does not repeat in a regular, fixed manner

and $d_j(i)$ is the Euclidean distance between the each pair of points after each discrete time step i (i.e. $i\Delta$t s) on the two trajectories (Figure 2-4C). Local divergence exponents (λ^*) were estimated from the slopes of linear fits to these exponential divergence curves in logarithmic scale:

$$y(i) = \frac{1}{\Delta t}\left\langle \ln\left[d_j(i)\right]\right\rangle \quad (2\text{-}4)$$

where $d_j(i)$ was the Euclidean distance between the j^{th} pair of initially nearest neighbors after i discrete time steps (i.e. $i\Delta t$ seconds) and $\langle \cdot \rangle$ denotes the average over all values of j (Rosenstein, Collins et al. 1993).

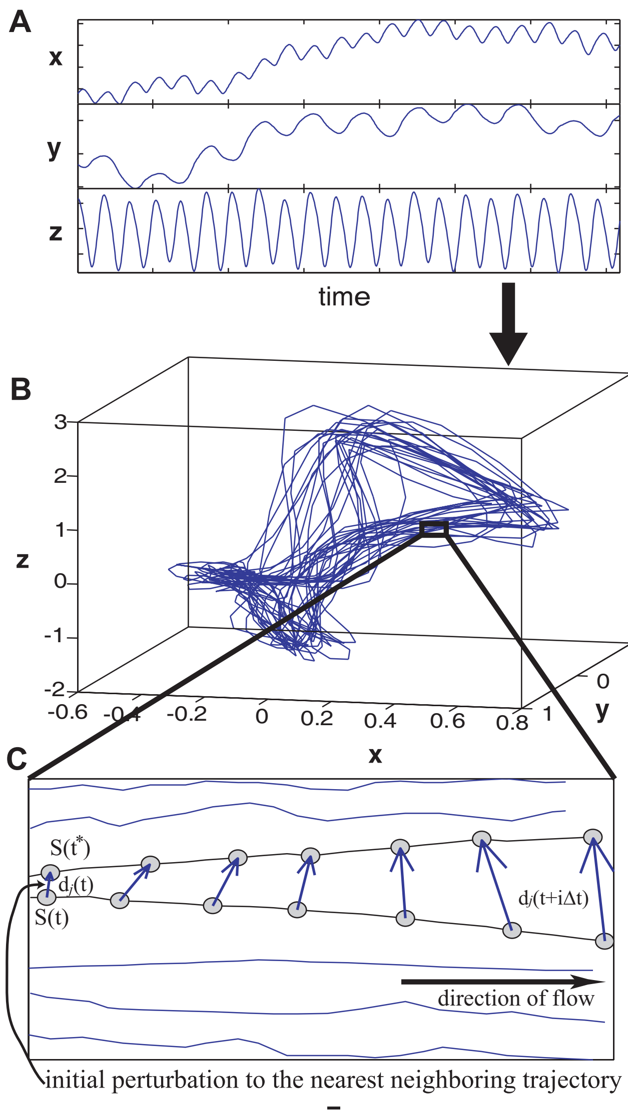
An adaptation of this approach quantifies the behavior of the entire mean divergence curve, rather than the slope of a specifically defined region. To quantify Lyapunov exponents, mean divergences are calculated on a logarithmic scale (mean log divergence), such that a linear line in a log scale indicates an exponential function.

However, because mean log divergence curves attained from human walking do not exhibit a distinct linear region, other methods have been developed to describe the entire curve (Kang and Dingwell 2006). Also, this method does not account for the strongly periodic[2] nature of walking, and examines only the overall trend in the data. This method cannot be used to distinguish different parts of the gait cycle. Also, existing methods to calculate λ_{max} from data compare a trajectory to its nearest neighbor, rather than to the limit cycle.

The perplexing finding from these studies was that all subjects exhibited a significant degree of local *in*stability during walking, even though no subject ever fell in these previous studies. This was likely due in part to inherent biological noise such as errors in motor commands that can also produce the same kind of sensitivity to initial conditions that local divergence exponents quantify (Theiler, Eubank et al. 1992;

[2] Periodic: having a regular interval, and recurring in the same manner after some fixed period

Dingwell and Cusumano 2000). This local instability may also result in part from small corrections made by the neuromuscular control system to smooth unintended irregularities during gait (Dingwell and Cusumano 2000; Haridas, Zehr et al. 2005). Thus, although these local divergence exponents do provide a rigorous quantifiable measure of how humans respond to local perturbations, it is not yet clear if they are directly related to a person's ability to walk without falling over, or if they are instead quantifying some other more subtle aspects of locomotor stability control.



(A) The original time series of raw data define the states of the system.

(B) These states are combined to form the system's trajectory in state space (only 3 states are shown here used for illustrative purposes).

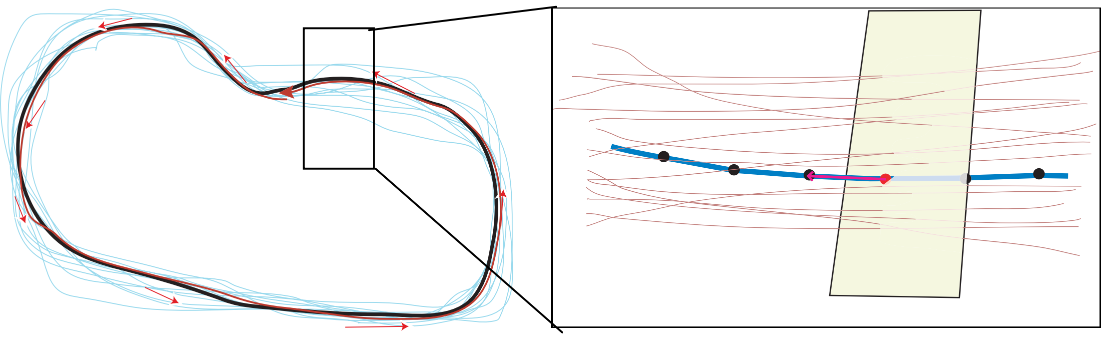
(C) Expanded view of a typical local region. A small perturbation moves the system at $S(t)$ to its closest neighbor $S(t^*)$. Local divergence is computed by measuring the Euclidean distances between the subsequent points, denoted $d_j(i)$. The local dynamic stability of the system is defined by how quickly the two trajectories diverge away from each other.

Figure 2-2. Schematic Representation of State-space Construction and Local Dynamic Stability Analysis for Estimating λ_{max}.

6.6 Floquet Theory and Orbital Stability

A second, rigorously defined approach for quantifying local stability exists for examining periodic systems. "*Orbital stability*" can be quantified using Floquet multipliers (Nayfeh and Balachandran 1995), which quantify how the system's states respond to local perturbations discretely *from one cycle to the next* at a single point during the cycle (e.g., heel strike). They quantify how a perturbation at cycle k (or stride k) will change after going through one full cycle (or stride). In other words, they track whether the effects of perturbation at a particular stride has increased or decreased by the next stride.

(a) (b)

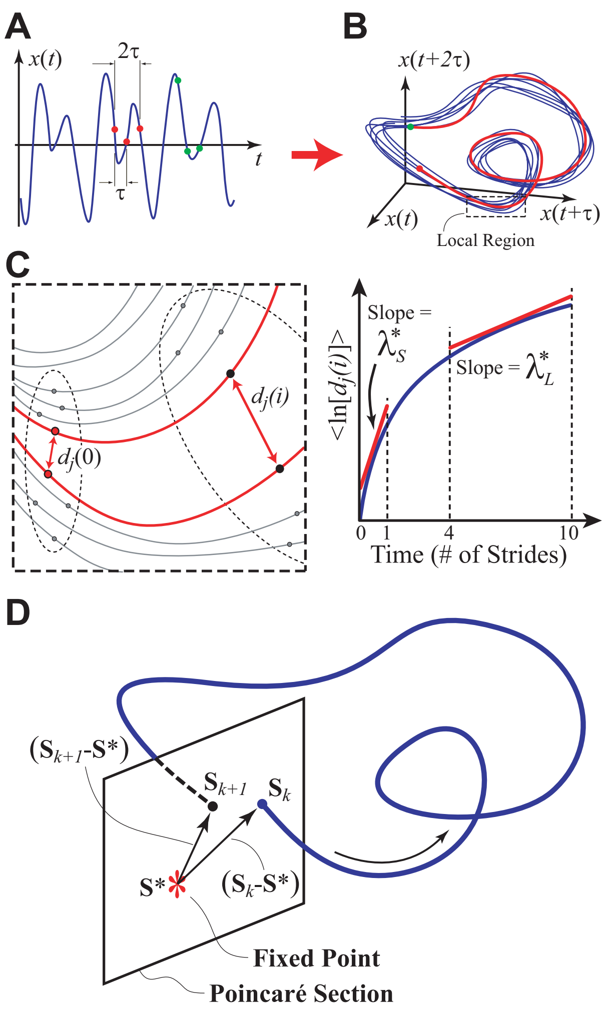


(a) A diagram of the state space. The thick black line represents the average cycle, or the attractor cycle.

(b) Once we take a "slice" of the state space, we can define the Poincaré section.

Figure 2-3. Illustration of the State Space.

This analysis is done on a particular part of the limit cycle, called the Poincaré section of the limit cycle (Figure 2-3). The trajectories of cycle k and cycle k+1 both intersect the Poincaré section. The relationship of cycle k to k+1, each relative to the limit cycle, or the mapping of cycle k to k+1 at a particular part of the cycle, is called the Jacobian matrix, whose eigenvalues are the Floquet multipliers. Similar to Lyapunov exponents, different state variables are affected in different ways, and the largest Floquet multiplier defines the stability of the system. A Floquet multiplier greater than 1 indicates that a perturbation will increase by that factor at the next cycle, and the perturbation will become greater and greater, indicating instability. A Floquet multiplier less than 1 indicates that a perturbation will decrease by that amount by the subsequent cycle, indicating stability.



A: Original time series data, $x(t)$, plotted as a function of time (arbitrary units). ***B***: Reconstruction of a 3-dimensional attractor for $x(t)$ such that $\mathbf{S}(t) = [x(t), x(t+\tau), x(t+2\tau)]$. The two triplets of points indicated in ***A*** and separated by time lags τ and 2τ each map onto a single point in the 3D state space. Alternatively, the physical variables can be used to construct the state space ***C***: Expanded view of a local section of the attractor shown in ***B***. An initial naturally occurring local perturbation, $d_j(0)$, diverges across i time steps as measured by $d_j(i)$. Local divergence exponents (λ^*) are calculated from the linear slopes of the average logarithmic divergence, $<\ln[d_j(i)]>$ (Eq. 2), of all pairs of initially neighboring trajectories vs. time. ***D***: Representation of a Poincaré section transecting the state space perpendicular to the system trajectory. The system state, $\mathbf{S}_k$, at stride k evolves to $\mathbf{S}_{k+1}$ one stride later. The Floquet multipliers quantify whether the distances between these states and the system fixed point, $\mathbf{S}^*$, grow or decay on average across many cycles. (adapted from Dingwell and Kang 2007)

Figure 2-4. Schematic Representation of Lyapunov (local) and Floquet (orbital) Dynamic Stability Analyses.

Floquet theory has been used extensively to assess stability in walking robots (Garcia, Chatterjee et al. 1998; Kuo 1999), but its application has been limited in human experiments. Human walking was found to be orbitally stable (Hurmuzlu and Basdogan 1994), but post-polio patients were less stable (higher Floquet Multipliers) than healthy controls (Hurmuzlu, Basdogan et al. 1996). Fall prone elderly exhibit larger Floquet multipliers at heelstrike (Granata and Lockhart 2006), while diabetic neuropathy and slower walking speeds do not affect orbital stability (Dingwell, Kang et al. 2007).

6.7 Local Stability along the Limit cycle

Building on both of these methods, more recent work describes mathematical limit cycles that can exhibit locally *un*stable regions and still remain orbitally stable (e.g., Figure 2-5) (Ali and Menzinger 1999). Compared to Floquet multipliers that only provide a "stroboscopic" view of the system at one particular part of the limit cycle and ignore what happens at other parts of the cycle, the local stability method proposed by Ali and Menzinger tracks different behavior along the limit cycle. This is also unlike Lyapunov exponent methods for data that can only give overall local stability trends of the system.

As stated in section 5, reflex modulation during walking suggests that perturbations are handled differently during different parts of the gait cycle suggesting that stability may vary as well throughout the gait cycle. Although the Ali and Menzinger method shows potential to yield more information, it has yet to be adapted to study human movement.

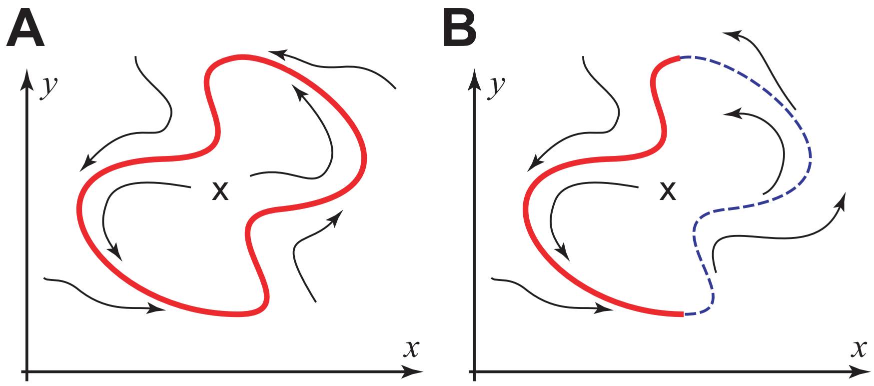


Figure 2-5. Limit Cycles with Locally Unstable Portions

A: Schematic representation of an orbitally stable limit cycle that is also locally stable everywhere along the limit cycle. Being locally stable everywhere guarantees that the limit cycle must also be orbitally stable. ***B***: Representation of an orbitally stable limit cycle composed of both locally stable (solid line) and locally unstable (dashed line) regions. Trajectories that veer away from the limit cycle in the locally unstable regions are then "drawn back" toward it again in the locally stable region. (Adapted from Ali and Menzinger 1999).

6.8 Summary and Next Steps

These stability measures in the literature come from the field of engineering dynamics. They quantify a response of the system to perturbations, and describe whether the system will return toward the previous trajectory or the limit cycle, or keep moving away. Each of them describes slightly different aspects of the system dynamics. Table 1 summarizes their use and characteristics.

Table 1. Summary of Existing Stability Measures Based on Dynamics

	Lyapunov Exponent/ Mean Divergence	**Floquet Multiplier**	**Ali and Menzinger local stability**
Perturbation size considered	Very small – nearest neighbors only	All sizes considered	Designed for small perturbations
Compares the current trajectory to:	A nearby trajectory	Limit cycle and the next cycle	Limit cycle
Applicability	Any time series data – has led to misuse in the literature without the proper understanding	Cyclic or periodic systems. Can be applied to any part of the gait cycle	Cyclic or periodic systems. Can be applied to any part of the gait cycle
Weaknesses	Current formulations do not separate different parts of the gait cycle	"Stroboscopic" – Ignores what happens during the rest of the cycle	For theoretical systems: No formulation exists to analyze recorded data
Effects of Stochastic elements	Appearance of local instability	Tolerates small amounts	unknown

To quantify the stability of walking in these proposed studies, specifically to test if different phases of the gait cycle, stability needs to be quantified comprehensively throughout the gait cycle. Existing methods of calculating Lyapunov exponents are not designed to separate the different parts of the gait cycle. Floquet multipliers also do not take into account behavior at other parts of the gait cycle. The Ali and Menzinger local stability method overcomes this limitation, by tracking the system behavior throughout the gait cycle, but it has not been implemented in a form to analyze data.

One limitation of these methods is that these measures (Lyapunov exponents, Floquet multipliers, Ali and Menzinger methods) were designed to study only deterministic systems that are specified as equations, and not for systems that have any noise or other stochastic components. However, human movement does not follow such exact mathematical specifications: one stride is never quite the same as another stride. Human movement exhibits natural variability, and biological noise is inherent in motor control (Harris and Wolpert 1998).

These “noise” or stochastic behaviors in biological systems seem to be the cause of previous observations of local instability in human walking, even when walking also exhibits simultaneous orbital stability. An explanation for this is that the nervous system allows for some amount of natural variability and instability, but it corrects larger perturbations (Dingwell and Kang 2007). In order to account for this stochastic behavior, a new way of quantifying stability is needed. The Ali and Menzinger method may help, but much work is needed before it can be used to study human walking.

Chapter 3: Data Collection and Analyses

This chapter describes the methodology used to collect the dataset used in all four studies described in chapters 4-7. Also, the analyses common to all four studies are described. Any methodology specific to each study is described in chapters 4-7.

SUBJECTS

Eighteen young healthy adults, age 18 to 28, and 18 older adults, age 65 to 85, were recruited. Recruitment took place by posting flyers on bulletin boards in Bellmont Hall and other nearby building on The University of Texas at Austin campus. Older adults were also recruited through a newspaper advertisement and through word of mouth. A health-history questionnaire was used to screen the subjects (Appendix A). Data from one young subject were discarded due to technical problems.

Table 2. Subject Characteristics

	Young adults	Older Adults
Sex ratio (M/F)	12/5†	12/6
Age (years)	23.3 ± 2.6	72.1 ± 6.0
Height (m)	1.73 ± 0.094	1.70 ± 0.104
Weight (kg)	71.1 ± 9.86	73.2 ± 12.3
Body Mass Index (BMI)	23.5 ± 1.7	25.4 ± 3.2
Preferred Walking Speed (m/s)	1.30 ± 0.10	1.29 ± 0.15
PWS Range (m/s)	1.16 – 1.56	0.93 – 1.52
Non-dimensionalized PWS (Hof 1996)	0.438 ± 0.037	0.436 ± 0.052
Composite Strength score*	3.07 ± 3.71	-4.29 ± 2.35
Composite ROM score*	1.89 ±1.81	-2.03 ± 2.78

† Reflects the number after one young subject data was discarded
* indicates $p < 0.0001$

INSTRUMENTATION

Health-history questionnaire

A 19-question questionnaire was used to screen potential study subjects. (Appendix A). The questions asked whether the subject was taking certain medications, had any recent lower-extremity injury, or other medical conditions that may influence the results of this study. Subjects using medications that can affect the nervous system or those who have had recent injuries to the lower extremities, congenital or acquired neurological injuries or deficits were excluded from the study.

Manual muscle tester

A hand-held dynamometer, Lafayette Manual Muscle Tester Model 01163 (Lafayette Instrument Company, Lafayette IN) was used to test muscle strength. As a class I medical device, it does not require a user calibration. It has an accuracy of ±1%, and a resolution of 0.2 kgf (1.96 N). One individual trained to operate this instrument assessed all subjects. A pilot study was conducted to quantify intra- and inter-session reliability. Inter-class correlation type (2,1) of 0.85 was attained.

Motion Capture System

A Vicon 612 system (Oxford Metrics, UK) with eight (8) MCam-1 cameras was used to collect kinematic data. Kinematics were collected at 60Hz from each camera, at 1 megapixel resolution. With the experiment setup shown (Figure 3-1), measurement residuals (expected RMS measurement error) between 0.5 and 1.0 mm were obtained.

Vicon Workstation software build 140 was used for collection and export of the raw kinematic and analogue data.

Treadmill

Subjects walked on a Woodway Desmo-S treadmill (Woodway, Waukesha, WI). This treadmill is capable of 0-15% incline, and speeds from 0 – 12.5 mph. This treadmill was chosen for its direct-servo drive that minimizes intra-stride variations in treadmill speed. As long as belt speed is constant and the walking surface is rigid, treadmill walking is mechanically identical to overground walking (van Ingen Schenau 1980).

PROTOCOL

First, informed consent was obtained. Second, preferred walking speed was determined, which also served as a warm-up. Third, passive range of motion was measured, and served as stretching. Fourth, isometric strength of leg muscles was measured. Finally, the subjects walked on the treadmill while their motion and muscle activity was recorded.

Preferred walking speed

Preferred self-selected walking speed of the subject was determined using a published protocol (Dingwell and Marin 2006). As the subject was walking, the treadmill speed was increased gradually from a slow speed until the subject noted that the speed felt a bit faster than the subject's comfortable walking speed. Then, the treadmill speed was decreased gradually from a high speed (beyond the first speed noted by the subject by about 1 mph but without causing the subject to break into a running gait), until the subject noted that the speed felt a bit slower than his or her comfortable walking speed. This process was repeated two more times, and the 6 speeds noted by the subject were averaged to determine preferred walking speed (PWS).

Joint Range of Motion

Joint range of motion was measured in all subjects to account for any joint range of motion related differences in the two groups. This allowed the use of joint range of motion as a covariate when comparing final dependent measures between the groups. Range of motion was measured using a goniometer using guidelines as described below developed from (Smidt 1994) with consultation with Ann Newstead, PT. Each joint was measured once, and both legs were measured.

Hip:

The subject lay supine on the table. The femur segment was defined from the lateral condyle of the femur to the greater trochanter. The trunk was defined from the greater trochanter, along the length of the trunk, parallel to the table. The subject started from the knee extended, and brought his or her knee to the chest, using the contralateral hand to stabilize the leg. The contralateral leg was kept straight. The subject was instructed to relax and not use other muscles to "help" the measurement, except for the hands. The tester bent the leg at the hip as far as it can go without being uncomfortable, and measured hip angles at each end. Two numbers were recorded: maximum extension angle (~0°) and maximum flexion angle (~120°). Hyperextension was not recorded. The range of motion was calculated as the difference between these 2 angles.

Knee:

The subject lay supine on the table. The thigh segment was defined from the lateral condyle of the femur to the greater trochanter. The shank segment was from the lateral condyle to the lateral fibular head. The subject started with the leg extended, and was asked to bring the knee to the chest, and use the contralateral hand to stabilize the leg. The tester flexed the leg at the knee as far as it would go without causing discomfort. Two angles were recorded: maximum extension angle (~0°) and maximum flexion angle (~120°). Hyperextension was not recorded. The range of motion was caculated from the difference between these 2 angles.

Ankle:

The subject lay supine with the feet dangling off the table. The shank segment was defined from the lateral condyle of the femur to the lateral fibular head. The foot segment was defined from the lateral head of the 5th metatarsal to lateral malleolus. Neutral position was defined when the foot is perpendicular to the fibula. Neutral position was defined as the foot at 90 degrees to ankle. From that position, the tester recorded: maximum dorsiflexion (0°-20°) and maximum plantarflexion (0°-45°). The range of motion was calculated from adding these angles together.

Strength testing protocol using Hand-held Dynamometry

Leg muscle strength also was recorded to be used possibly as a covariate when comparing the two groups. The following protocol adapted from Smidt 1994 was used. Each measurement was performed twice, with a minimum 30 seconds of rest in between. The tests were conducted in the following order: hip flexors, knee flexors, knee extensors, hip extensors, plantaflexors, then dorsiflexors. Both the force produced, and the joint moment-arm length was measured using a measuring tape.

Hip flexors:

Subjects sat with hip and knee joint at 90 deg flexion, grasping the edge of the table, as the lower legs hung off the table. The force measurement point was at the anterior surface of distal thigh, with the subject pushing up, and the operator pushing down with as little motion as possible. The moment arm was measured from the greater trochanter to the point of force measurement.

Hip Extensors:

The subject was supine, hip at 50 deg flexion, with full knee extension. The pelvis was held down using a strap attached to the table for stability. The measurement point was at the posterior surface of the distal shank, with the subject pushing down, and the operator pushing up. The subject was instructed to keep the leg straight. The moment-arm length was measured from the greater trochanter to the point of force measurement.

Knee Extensors

The subject sat with the knee at 90 degrees. The measurement point was the anterior surface of the distal shank, with the subject pushing forward, and the operator pushing backward. The moment-arm length was measured from the lateral epicondyle of the femur to the point of force measurement.

Knee Flexors

The subject sat with the knee at 90 degrees. The measurement point was at the posterior surface of the distal shank, with the subject pushing backward, and the operator pushing forward. The moment-arm length was measured from the lateral epicondyle of the femur to the point of force measurement.

Ankle Plantarflexors

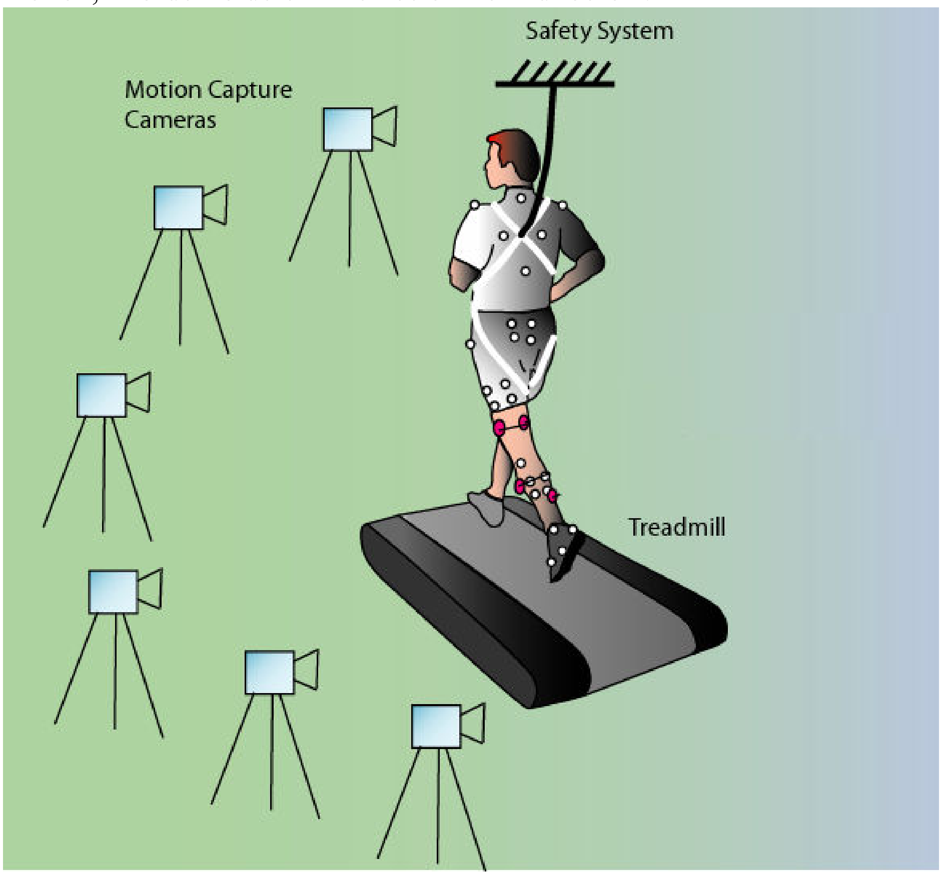
The subject was supine, with the leg extended, and the ankle at neutral. The feet and ankles were off the table. The operator held down the pelvis and the distal shank for stability using straps. The measurement point was the plantar surface of the foot, at the metatarsal phalangeal joints 1-3 (ball of the foot), with the subject pushing toes away from his/her head, and the operator pushes toward the head. The moment-arm length was measured from the lateral malleolus of the fibula to the point of force measurement.

Ankle dorsiflexors

The subject stood leaning against a supporting surface, with most of his or her weight on the foot to be tested. The measurement point was the dorsum of the foot, at the metatarsal phalangeal joints 1-3. The subject pushed the toes upward, and the operator pushed downward, making sure that the heel stayed in contact with the ground while most of the forefoot was off the ground.

Kinematic and EMG Measurements

Kinematics were measured at 60 Hz sampling frequency. A set of 31 reflective markers (14 mm size) was used to track motions of the subject. Six markers were placed on the trunk: left and right acromion, spinal processes of C7, T10, and the spines of bilateral scapulae; 5 on the pelvis: 1 on a wand on left anterior-superior iliac spine (ASIS), and a 4-marker cluster on the sacrum; a 4-marker cluster on the left thigh and 1 on the lateral epicondyle of the left femur (“knee”); a 4-marker cluster on the left shank, 1 on the left lateral malleous ("ankle"); and 5 on the left shoe: one near (as close as possible) to the head of the 2nd phalanx, 1 near the head of the 5th metatarsal, 1 on the dorsum of the foot, one inferior to the "ankle" and one near the calcaneous of the foot, all on the left side. An additional five markers were placed on the right foot to track its motion, in order to determine heelstrike and toe-off.



The subjects walked on a treadmill while their motion was recorded. White circles represent the reflective markers. Red circles represent EMG electrodes. Coordinate system: x = Anterior-posterior; y = medial-lateral; z = vertical

Figure 3-1. Experimental Setup

Muscle activation patterns were measured using surface electromyography (EMG). The bipolar electrodes (DE 2.1, Delsys, Boston MA) were placed on 4 muscles of the left leg: *vastus lateralis*, hamstrings, medial head of *gastrocnemius*, and *tibialis anterior* according to the SENIAM conventions (Konrad 2005). The "hamstring" electrode was placed superficial to semitendinosus muscle, but due to the proximity of biceps femoris and semimembranosus muscles, it would be incorrect to refer to it as a signal from other muscles. The skin was prepared by shaving and using alcohol to remove excess dead skin and oils. EMG signals were amplified 1000x and sampled at 1080 Hz.

Walking Protocol

There were a total of 10 walking trials, 5 minutes each. Subjects were instructed to walk while looking ahead, and avoid extraneous movements such as talking, scratching the nose, etc. The 10 trials consisted of 5 different speeds, and 2 trials at each speed. The speeds were the Preferred Walking Speed (PWS) as determined from the protocol above, PWS ± 10%, and PWS ± 20%. The order of presentation was pseudo-randomized in the following manner (Table 3). The order of presentation was designed such that all 5 speeds were presented in the first 5 trials, in case a subject did not wish to continue, and the high speeds (PWS + 10%, +20%) were not presented back-to-back, in order to prevent fatigue. Between each walking trial, the subjects were allowed to rest as much as desired, but at least 2 minutes.

Table 3. Order of Presentation of Speeds

	trial # - Order of presentation									
Subject #	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
1	2	5	3	1	4	2	3	5	1	4
2	5	3	1	4	2	4	2	3	5	1
3	3	1	4	2	5	1	4	2	3	5
4	1	4	2	5	3	5	1	4	2	3
5	4	2	5	3	1	3	5	1	4	2
6	3	2	5	1	4	3	4	2	5	1
7	2	5	1	4	3	1	3	4	2	5
8	5	1	4	3	2	5	1	3	4	2
9	1	4	3	2	5	2	5	1	3	4
10	4	3	2	5	1	4	2	5	1	3
11	3	2	4	1	5	2	4	1	3	5
12	2	4	1	5	3	5	2	4	1	3
13	4	1	5	3	2	3	5	2	4	1
14	1	5	3	2	4	1	3	5	2	4
15	5	3	2	4	1	4	1	3	5	2
16	2	4	3	1	5	3	4	1	5	2
17	4	3	1	5	2	2	3	4	1	5
18	3	1	5	2	4	5	2	3	4	1

1 = 80% of PWS, 2 = 90% of PWS, 3 = PWS, 4 = 110% of PWS, 5 = 120% of PWS

ANALYSIS

Data Processing

Three-dimensional (3D) Marker positions were reconstructed from the camera data using Vicon Workstation software. Any gaps in the marker trajectories up to and including 6 frames were filled using a built-in spline function in Vicon Workstation. Any additional gaps were filled using a custom MATLAB routine that calculated the likely position of a missing marker based on the location of other markers using rigid-body assumptions. Hip joint center was estimated using a custom MATLAB routine using the markers from the thigh segment and the pelvis segment. Because this calculation was over-determined, optimization was used to determine a location on the pelvis that was equidistant from both thigh and pelvis markers.

Gait Event Determination

Heelstrikes and toe-offs were determined from the kinematic data of both feet. An heelstrike was defined as the point where the heel marker of the forward foot was at its most forward point within a gait cycle. Toe off was defined as the point where the toe marker was at its rear-most point on the treadmill. From this information, stride Time was defined as time from one heel contact to the next ipsilateral heel contact. Step Length was defined as length between the heel and the contralateral heel at each heel contact in the anterior-posterior direction. Step width was defined as the length between the heel and the contralateral heel at each heel contact.

Segment and Joint Angles

Segment angles and joint angles in 3D were calculated, using rotation matrices derived from the marker positions on each segment. In conformity with common clinical rotation sequence convention, and implemented in many gait laboratory software packages such as Vicon Clinical Manager and Peak Motus, the tilt-obliquity-rotation (TOR) sequence was used, which refers to flexion, abduction, and internal rotation in anatomical terms (Grood 1983). This Cardan rotation sequence convention relates the rotation of the distal segment to the proximal segment. The first axis of rotation is fixed on the proximal segment, and the third axis of rotation is fixed on the distal segment, and the 2nd axis "floats" (Baker 2001). Other conventions have been proposed, such as rotation-tilt-obliquity, as used by Motion Analysis corporation, and the rotation-obliquity-tilt for pelvis motion, but these are not used as commonly (Baker 2001).

In the data collection setup, anterior-posterior direction was in X, medial-lateral direction in Y, and vertical direction in Z. Using the tilt-obliquity-rotation convention, the rotations occurred in Y-x'-z'' order. Rotation matrices, $\Re(t)$, were computed from the movements of the markers on each segment with respect to the mid-stance anatomical position using a singular value decomposition method briefly described here (Söderkvist and Wedin 1993).

Given a segment at position A and B with n multiple markers, where each marker is represented by a column of x, y, and z coordinates,

$$\mathrm{A} = \begin{bmatrix} x_1 & x_2 & x_3 & & x_n \\ y_1 & y_2 & y_3 & \ldots & y_n \\ z_1 & z_2 & z_3 & & z_n \end{bmatrix} \qquad \mathrm{B} = \begin{bmatrix} X_1 & X_2 & X_3 & & X_n \\ Y_1 & Y_2 & Y_3 & \ldots & Y_n \\ Z_1 & Z_2 & Z_3 & & Z_n \end{bmatrix} \tag{3-1}$$

the mean marker positions are subtracted from both A and B, then they are multiplied together:

$$\mathrm{C} = \mathrm{BA}^{\mathrm{T}} = \begin{bmatrix} X_1-\overline{X} & X_2-\overline{X} & X_3-\overline{X} & \\ Y_1-\overline{Y} & Y_2-\overline{Y} & Y_3-\overline{Y} & \ldots \\ Z_1-\overline{Z} & Z_2-\overline{Z} & Z_3-\overline{Z} & \end{bmatrix} \begin{bmatrix} x_1-\overline{x} & y_1-\overline{y} & z_1-\overline{z} \\ x_2-\overline{x} & y_2-\overline{y} & z_2-\overline{z} \\ x_3-\overline{x} & y_3-\overline{y} & z_3-\overline{z} \\ & & \\ & \ldots & \end{bmatrix} \tag{3-2}$$

C, now a 3x3 matrix, can be decomposed into *P*, diagonal matrix of singular values, and *Q* using singular value decomposition:

$$C = P\begin{bmatrix} \sigma_1 & 0 & 0 \\ 0 & \sigma_2 & 0 \\ 0 & 0 & \sigma_3 \end{bmatrix} Q^T \tag{3-3}$$

The rotation matrix for this segment can be calculated from *P* and *Q*:

$$\Re_{seg} = P\begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & \det(PQ^T) \end{bmatrix} Q^T \tag{3-4}$$

This rotation matrix defines the 3D rotation of this segment from position A to position B. By itself, it describes the rotation of a segment to itself, or *segment* angles. To calculate joint angles between two segments, this process is repeated for an adjoining segment. Then the rotation matrix that defines *joint* angles was calculated thusly:

$$\Re_{\text{joint}} = \left[\Re_{seg1}\right]^{-1}\left[\Re_{seg2}\right]$$

Once the rotation matrix was calculated, the segment or joint angles were defined according to the Cardan Y-x'-z'' convention:

$$\Re(t) = \begin{bmatrix} \sin\gamma\sin\alpha\sin\beta + \cos\gamma\cos\beta & \sin\gamma\cos\alpha & \sin\gamma\sin\alpha\cos\beta - \cos\gamma\sin\beta \\ \cos\gamma\sin\alpha\sin\beta - \sin\gamma\cos\beta & \cos\gamma\cos\alpha & \sin\beta\sin\gamma + \cos\gamma\sin\alpha\cos\beta \\ \cos\alpha\sin\beta & -\sin\alpha & \cos\alpha\cos\beta \end{bmatrix} \quad (3\text{-}5)$$

From here, the Cardan angles were calculated (Greenwood 1988):

Flexion/Extension (about Y-axis): $\beta(t) = \sin^{-1}\left(\dfrac{\Re_{3,1}}{\cos\alpha}\right)$ (3-6)

Adduction/Abduction (about x'-axis): $\alpha(t) = -\sin^{-1}\left(\Re_{3,2}\right)$ (3-7)

Internal/External Rotation (about z''-axis): $\gamma(t) = \sin^{-1}\left(\dfrac{\Re_{1,2}}{\cos\alpha}\right)$ (3-8)

Linear velocities of the trunk COM relative to global coordinates were quantified by using 3-point difference formula to calculate the numerical derivative. Trunk velocities were used instead of trunk positions, to reduce the non-stationarity in the data (Dingwell and Marin 2006). Trunk COM was estimated using anthropometric tables (Winter 1990).

EMG Processing

The recorded EMG signals were bandpass filtered (passband 20-300Hz) and demeaned. EMG was normalized to the peak amplitude during the preferred walking speed trial. Signals were then rectified, and low-pass filtered using the Hamming convolution filter with the averaging window of 100 ms. This generated the smoothed linear envelopes.

Stability Calculations

The methods to quantify local dynamic stability and orbital stability are described here. These calculations were done on a state-space of interest, defined more in detail in Chapters 5-7.

State Space Description of Segments

The perturbation response during standing and walking can be estimated by examining the trajectories of the system's movements in state space. When properly defined, the state variables of a system fully describe its behavior. For example, a mechanical system consisting of a single point mass with 1 degree of freedom can be described by a 2^{nd} order differential equation:

$$F(t) = k\mathbf{x} + b\dot{\mathbf{x}} + m\ddot{\mathbf{x}} \qquad (3\text{-}10)$$

where $\mathbf{x}$, $\dot{\mathbf{x}}$, and $\ddot{\mathbf{x}}$ are position, velocity and acceleration, *k*, *b*, and *m* are system parameters (i.e. stiffness, damping, and mass), and *F*(*t*) is the external force applied to the system. This equation constrains the acceleration to be a function of position and velocity. Therefore, the system's position and velocity can be used to define the two state variables that are sufficient to fully describe the dynamics of the system, $S(t) = [\,\mathbf{x},\ \dot{\mathbf{x}}\,]$ or $S(t) = [\,\dot{\mathbf{x}},\ \ddot{\mathbf{x}}\,]$ (Pai and Patton 1997). An extended 3-dimensional rigid body has three translational plus three rotational degrees of freedom. The dynamics of this 2nd order system can therefore be fully described in terms of these 6 degrees of freedom [x y z θ ϕ ψ] and their time derivatives $[\dot{x}\ \dot{y}\ \dot{z}\ \dot{\theta}\ \dot{\phi}\ \dot{\psi}]$, or using 2nd derivatives $[\ddot{x}\ \ddot{y}\ \ddot{z}\ \ddot{\theta}\ \ddot{\phi}\ \ddot{\psi}]$ thus forming a 12-dimensional state space such as these:

$$S(t) = [x, y, z, \dot{x}, \dot{y}, \dot{z}, \theta, \phi, \psi, \dot{\theta}, \dot{\phi}, \dot{\psi}] \qquad (3\text{-}11)$$

$$S(t) = [\dot{x}, \dot{y}, \dot{z}, \ddot{x}, \ddot{y}, \ddot{z}, \dot{\theta}, \dot{\phi}, \dot{\psi}, \ddot{\theta}, \ddot{\phi}, \ddot{\psi}] \qquad (3\text{-}12)$$

A state-space also can be reconstructed consisting of the velocity variables and their time-delayed copies (Packard, Crutchfield et al. 1980; Takens 1981).

$$\begin{aligned} S(t) = [&\dot{x}(t) \quad \dot{y}(t) \quad \dot{z}(t) \quad \dot{\theta}(t) \quad \dot{\phi}(t) \quad \dot{\psi}(t) \ldots \\ &\dot{x}(t-\tau_1) \quad \dot{y}(t-\tau_2) \quad \dot{z}(t-\tau_3) \quad \dot{\theta}(t-\tau_4) \quad \dot{\phi}(t-\tau_5) \quad \dot{\psi}(t-\tau_6)] \end{aligned} \qquad (3\text{-}13)$$

where the time delays (τ) were calculated from the first minimum of the average mutual information function of each time series [Abarbanel 2001].

For each segment (thorax, pelvis, thigh, shank, foot), the linear motions were defined from the linear excursions of the center of mass (COM) estimates using standard anthropometry methods (Winter 1990). The state variables and time variables were non-dimensionalized in order to make meaningful comparisons between subjects and groups, using scaling factors described in the literature (Hof 1996). Lengths were be normalized by leg length (l); Time was normalized by $\sqrt{l/g}$; Velocity was normalized by $\sqrt{lg}$, where $g = 9.81$ m/s^2.

Segment rotation angles relative to global coordinate system were calculated using tilt-obliquity-rotation sequence as above. From these values, the linear and angular velocities were calculated using the 3-point difference formula. This defined the 12-D state space for each segment. Angular velocities were calculated from the time-derivatives of Cardan angles. They were not transformed to be in relation to global (lab) coordinate system, because this process made them non-stationary.

Defining the Attractor

For calculating orbital stability, an orbit needs to be defined, with respect to which each stride will be compared. The exact nature of the attractor orbit (limit cycle, pseudoperiodic, strange/chaotic, etc.) or its mathematical behavior in the context of human walking is not known. However, over time human walking tends to follow the mean cycle, or the average stride. This suggests that the nervous system is trying to keep the locomotor pattern at that average stride. Therefore the attractor of the system was defined as the mean cycle. The mean cycle (MC) was defined by first normalizing each stride to 0-100% of the gait cycle, and averaging each state variable across strides.

$$MC_j(i) = \left\langle X_j(i) \right\rangle, i \in \{0 - 100\% \text{ gait cycle}\} \qquad (3\text{-}14)$$

where X_j is the jth state variable.

Mean Log Divergence and Local Dynamic Stability

The local stability of a system can be described by the response of that system to small perturbations. When a perturbation is introduced to the system moving through its state space, the system will be "bumped" to a nearby part of the state space. The system's new trajectory may converge back to the original trajectory, parallel it, or diverge away. The *rate* of divergence of neighboring state trajectories estimates local dynamic stability by measuring, on average, how quickly the system would diverge away from the original trajectory after being "bumped" over to a neighboring trajectory (Figure 2-4C). This divergence can be measured from the successive Euclidian distances between the points on the original and the "perturbed" trajectories (Rosenstein, Collins et al. 1993). Mean divergence quantifies the average rate of divergence between neighboring trajectories in state space. A steeper increase of mean divergence indicates that the system diverges faster from its original path in response to a small perturbation, and thus exhibits greater local dynamic *in*stability.

Mean divergence of the nearest neighbor trajectories was calculated using a previously published algorithm (Rosenstein, Collins et al. 1993) that was modified to use the state space trajectories defined above rather than delay-reconstructed state spaces from a single time-series. For each point $S(t)$ on the state-space trajectory, the nearest neighboring point $S(t^*)$ on an adjacent trajectory (excluding points on the same trajectory) was determined, forming the j^{th} pair of nearest neighbors. Euclidean distances between each pair of subsequent points on the two trajectories were then calculated. For this j^{th} nearest neighbor pair of $S(t)$ and $S(t^*)$, this formed a vector of the natural log of the Euclidean distances $d_j(i)$:

$$d_j(i) = \left\| S(t + i\Delta t) - S(t^* + i\Delta t) \right\|_2 \qquad (3\text{-}15)$$

and $d_j(i)$ is the Euclidean distance between the each pair of points after each discrete time step i (i.e. $i\Delta$t s) on the two trajectories. The neighboring trajectories were tracked for 30 seconds beyond the initial perturbation. This process was repeated for all initially neighboring points from the data set and the $d_j(i)$ for each pair of points were averaged to define the mean log divergence vector, $\langle \ln[d_j(i)] \rangle$, where $\langle \cdot \rangle$ denotes the arithmetic mean over all values of j. Divergence behavior was modeled as an exponential process, and the slope of the mean log divergence curve was used as a measure of exponential divergence (Dingwell and Marin 2006). Since the curve is not linear, slopes, or divergence exponents, were defined in two regions: 0-1 strides ("short-term" maximum finite-time Lyapunov exponent) denoted by λ^*_S, and 4-10 strides ("long-term" maximum finite-time Lyapunov exponent) denoted λ^*_L.

Orbital Stability

For the present study, orbital stability was quantified by calculating the Floquet Multipliers (FM) for the system (Nayfeh and Balachandran 1995; Dingwell and Kang 2007; Dingwell, Kang et al. 2007) based on well-established techniques (Hurmuzlu and Basdogan 1994; Hurmuzlu, Basdogan et al. 1996; Kuo 1999; Donelan, Shipman et al. 2004). Because Floquet theory assumes the system is strictly periodic, the state space data for each stride were first time-normalized to 101 samples (0% to 100%). This then allowed us to define 101 Poincaré maps (Figure 2-4D) for the system as:

$$\mathbf{S}_{k+1} = \mathbf{F}(\mathbf{S}_k) \quad (3\text{-}16)$$

where k was an index enumerating the individual strides and $\mathbf{S}_k$ denoted the system state (Eq. 1) for a single point in normalized time within each gait cycle. Attractor trajectories (mean cycle) as defined above correspond to single fixed points in each Poincaré map:

$$\mathbf{S}^* = \mathbf{F}(\mathbf{S}^*) \quad (3\text{-}17)$$

For our walking data, we defined the fixed points at each Poincaré section (*i.e.*, at each % of the gait cycle) by the average trajectory across all strides within a trial (see Appendix E for more details). Orbital stability at each Poincaré section was estimated by computing the effects of small perturbations away from these fixed points, using a linearized approximation of Eq. (4):

$$[\mathbf{S}_{k+1} - \mathbf{S}^*] \approx J(\mathbf{S}^*)[\mathbf{S}_k - \mathbf{S}^*] \quad (3\text{-}18)$$

where $J(\mathbf{S}^*)$ defined the Jacobian matrix for the system at each Poincaré section. The Floquet multipliers are the eigenvalues of $J(\mathbf{S}^*)$ (Hurmuzlu and Basdogan 1994; Hurmuzlu, Basdogan et al. 1996; Kuo 1999; Donelan, Shipman et al. 2004). Any deviation away from the fixed point is multiplied by FM by the subsequent cycle. Thus, for the attractor to be orbitally stable, these complex-valued FM must have magnitude < 1 (*i.e.*, they must all lie *inside* the unit circle). If any of the FM have magnitude > 1, the attractor is orbitally unstable. We computed the magnitudes of the maximum FM for each % of the gait cycle to determine how the orbital stability varied across the gait cycle. For statistical analyses, we extracted the largest of these maximum FM's ("Max FM") from across all Poincaré sections because this represented that instant during the gait cycle that was most unstable.

Statistics

For hypothesis testing, a repeated-measures analysis of variance (ANOVA) was used, with the following General Linear Model (Littell, Milliken et al. 1996).

$$y_{ijk} = \mu + \alpha_i + d_{ij} + \tau_k + (\alpha\tau)_{ik} + e_{ijk} \qquad (3\text{-}19)$$

Where:

i = 1, 2 (group)
j = 1 to J (subject)
k = 1 to 5 (speed, for Aims 1, 2, and 4)

y_{ijk} = the measurement at the k^{th} speed on the j^{th} subject assigned to the i^{th} group.
α_i = fixed effect of group
τ_k = fixed effect of speed
$(\alpha\tau)_{ik}$ = fixed effect of group*speed
$\mu + \alpha_i + \tau_k + (\alpha\tau)_{ik}$ = the mean measurement for group *i* at speed *k*, containing effects for group, speed, and the group* speed interaction.
d_{ij} = the random effect associated with the j^{th} subject assigned to group *i*.
e_{ijk} = the random error associated with the j^{th} subject assigned to the i^{th} group at speed *k*.

This model was used for all 4 studies, except in Aim 3, "k" referred to segments (trunk, pelvis, thigh, shank, foot) instead of speed.

Chapter 4: Separating the Effects of Aging and Walking Speed on Gait Variability

INTRODUCTION

Every year, over one-third of adults over age 65 fall. These falls represent the primary cause of accidental death in this population (Hornbrook, Stevens et al. 1994; Murphy 2000; Hausdorff, Rios et al. 2001). Increased gait variability has been shown to predict fall risk (Lord, Lloyd et al. 1996; Hausdorff, Rios et al. 2001). Even healthy older adults display greater gait variability than healthy young adults (Öberg, Karsznia et al. 1993; Öberg, Karsznia et al. 1994; Owings and Grabiner 2004), but the cause of this difference is not well understood. Older adults typically walk slower (Winter, Patla et al. 1990; Prince, Corriveau et al. 1997), and healthy young adults become more variable when they walk at slower speeds (Yamasaki, Sasaki et al. 1991; Dingwell and Marin 2006; Jordan, Challis et al. 2006). This suggests that increased variability observed in healthy older adults may be simply a result of slower walking speed. Alternatively, this greater variability may come from other factors related to aging, such as nervous or musculoskeletal deterioration, independent from slower walking. However, the speed-dependency of gait variability has not been characterized in older adults.

With fixed walking speeds on a treadmill, speed and variability in young adults display a quadratic relationship, where variability increases at speeds slower or faster than preferred (Yamasaki, Sasaki et al. 1991; Dingwell and Marin 2006; Jordan, Challis et al. 2006). However, this has not been demonstrated in older adults. Previous investigations of the effects of walking speed on gait variability in older adults used self-selected overground walking, where subjects were directed to walk "slow," "fast," etc. (Öberg, Karsznia et al. 1993; Öberg, Karsznia et al. 1994; Moe-Nilssen and Helbostad

2005). Although these approaches allow comparison among speeds, it is difficult to make comparisons between subjects or groups, since each subject walks at different speeds from other subjects. Conversely, interpolating variability at a fixed speed allows comparison between groups (Moe-Nilssen and Helbostad 2005), but ignores differences between individuals.

Older adults may exhibit a similar relationship between variability and walking speed as young adults, and thus their increased variability might be explained by their slower speed. Alternatively, the increased variability seen in older adults may exist regardless of speed, suggesting that it arises from other causes, such as loss of strength or flexibility. Additionally, changes in walking speed may increase variability even more in older adults than young adults.

To understand whether the greater gait variability in healthy older adults can be attributed to slower walking speed alone, we compared gait variability in both young and older adults across multiple controlled walking speeds using a treadmill. We aimed to determine if the increased variability in older adults was related to slower walking, or if other factors, specifically leg strength and flexibility contribute as well.

METHODS

Eighteen healthy older adults (age 65-85) and 17 height-, weight-, gender-matched young adults (18-28), participated after providing informed consent as approved by the University of Texas Institutional Review Board (Table 2). Subjects were recruited through advertising, and screened to exclude anyone who reported any history of orthopedic problems, recent lower extremity injuries, any visible gait asymmetries, or were taking medications that may have influenced their gait.

Subjects walked on a level treadmill (Desmo S model, Woodway USA, Waukesha WI) while wearing a safety harness (Protecta International, Houston TX) that allowed natural arm swing. First, individual preferred self-selected walking speed (PWS) was determined (Dingwell and Marin 2006). Subjects reported the limits of their PWS while the treadmill was slowly accelerated, then decelerated three times. These upper and lower limits were averaged to determine PWS. This allowed for treadmill acclimation and warm-up. Second, bilateral hip, knee, and ankle passive range of motion were measured using a goniometer. Bilateral isometric strengths (joint torques) of hip flexors, extensors, knee flexors, extensors, dorsiflexors and plantarflexors were measured using a hand-held dynamometer (Lafayette Instrument Company, Lafayette IN) using a protocol adapted from Smidt (Smidt 1994). Finally, subjects completed two 5-minute walking trials at five different speeds, wearing their own "walking" shoes. Speeds of 80, 90, 100, 110 and 120% of PWS were presented in a pseudo-randomized manner to avoid consecutive fast trials to prevent fatigue. Subjects rested at least 2 minutes between trials. Subjects were instructed to look ahead, avoiding extraneous movements while walking. Data from one trial each from one older subject and two young subjects were discarded due to technical problems. One older subject could not complete the 120%-speed trial, and this trial was also discarded.

Kinematics of 31 14-mm markers were measured using an 8-camera Vicon 612 system (Oxford Metrics, UK). Six markers were placed on the trunk (left and right acromion, spinal processes of C7, T10, and bilateral scapular spines); five on the pelvis (one on a wand on left ASIS, and a 4-marker cluster on the sacrum); a 4-marker cluster on the left thigh and one on the lateral epicondyle of the left femur; a 4-marker cluster on the left shank, one on the left lateral malleolus; and five on the left shoe (the head of the 2nd phalanx, the head of the 5th metatarsal, the dorsum of the foot, inferior to the fibula, and the calcaneous). Five additional markers were placed on the right foot to track heelstrike and toe-off (Figure 3-1).

Kinematics were recorded using Vicon Workstation 4.7 software, then other processing was done using MATLAB 7.04 (Mathworks, Natick MA). Gaps were filled using a custom routine using rigid-body assumptions. The location of the hip joint center was estimated using a custom optimization routine using the markers from the thigh and pelvis segments. The routine found a point fixed to the pelvis reference frame that was a fixed distance from any given marker on the two segments throughout the entire trial. The optimization minimized J, the sum of the variance over the entire trial, of the distance between the estimated joint center and the each marker (Eqn. 4-1):

$$J = \sum_n \operatorname*{var}_t \left[\left\| X_n(t) - EJC(t) \right\| \right] \qquad (4\text{-}1)$$

where n = marker number, $X_n(t)$ = position of marker n at time t, and EJC is the estimated joint center. Trunk center of mass was calculated using anthropometric tables (Winter 1990).

A heelstrike was defined as the point where the heel marker of the forward foot was at its most forward point within a gait cycle. Toe off was defined as the point where the toe marker was at its rear-most point on the treadmill. From these gait events, stride time was calculated as the time from one heel contact to the next ipsilateral heel contact. Step Length was defined as the length between the heel and the contralateral heel at each heel contact in the anterior-posterior direction. Step width was defined similarly in the mediolateral direction.

Three-dimensional joint angles at the hip, knee, and ankle joints were calculated using rotation matrices derived from the marker positions on each segment. The tilt-obliquity-rotation (TOR) sequence was used, which refers to flexion, abduction, and internal rotation in anatomical terms (Grood and Suntay 1983). During data collection, X defined the anterior-posterior direction, Y was medial-lateral, and Z was vertical. Rotation matrices were computed from the movements of the markers on each segment with respect to the mid-stance anatomical position using a singular value decomposition method (Söderkvist and Wedin 1993). From the rotation matrix, the 3D angles were defined according to the Cardan Y-x'-z'' (TOR) convention. Trunk segment angles were calculated relative to the laboratory reference frame. Linear velocities of the trunk COM were calculated by using the standard 3-point difference formula. Trunk velocities were used instead of positions to reduce the non-stationarity in the data (Dingwell and Marin 2006).

Composite strength and range of motion (ROM) scores were defined using Principal Components Analysis (Dingwell and Cavanagh 2001). A composite "Strength" was defined as the 1st principal component, the linear combination of the standardized isometric joint torque measurements that explained the most variance in the data. A composite "ROM" was defined similarly.

The data for each stride during walking were normalized to 0-100% gait cycle. Means and standard deviations of the joint angles and trunk motions were calculated at each percentage of gait cycle. To determine the variability of these measures over the entire gait cycle, the MeanSD of each variable was determined (Eqn. 4-2; Fig 4-1) (Dingwell and Marin 2006):

$$MeanSD = \langle SD(i) \rangle_i,\ i \in \{0 - 100\%\ \text{gait cycle}\} \qquad (4\text{-}2)$$

where $SD(i)$ indicates the standard deviation of a measure at ith % gait cycle, and $\langle\ \rangle_i$ denotes the average over all i. Standard deviations of spatio-temporal measures and MeanSD measures were compared between age groups and speeds using a 2-factor repeated-measures ANOVA using SPSS 14 (SPSS, Chicago IL). These analyses were repeated as ANCOVAs, where the composite strength and ROM scores were included as covariates. To reduce type II error, a Bonferroni correction of $\alpha = 0.05 \div 18 \approx 0.0028$ was used.

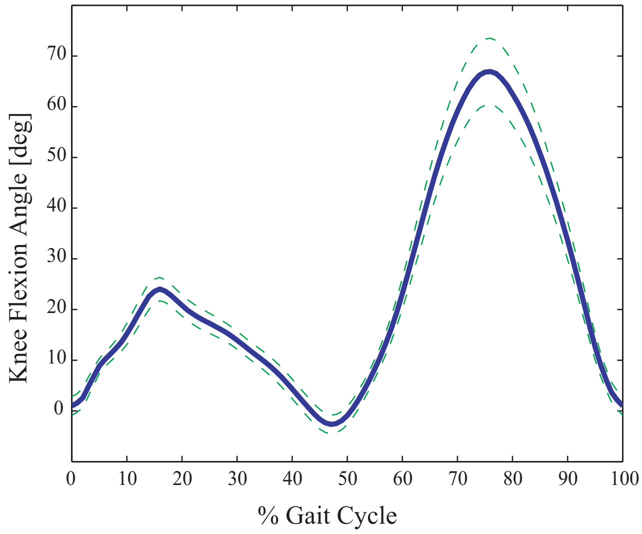


Figure 4-1. MeanSD Calculation

Blue line is the inter-stride mean knee flexion angle. Green dashed lines indicate ± 1 S.D. MeanSD is equivalent to average thickness of the green band around the blue line.

RESULTS

The preferred walking speeds (PWS) of older adults were no different from young adults, but exhibited a slightly wider distribution (all subjects: 1.29 ± 0.13 m/s, $p = 0.86$; Table 2). Older adults had lower strength and ROM scores ($p < 0.001$). Older adults exhibited significantly greater variability at all speeds for trunk roll ($p < 0.0003$). Step length ($p < 0.005$), and to lesser extent, stride time ($p < 0.02$), and trunk pitch ($p < 0.03$) exhibited similar trends (Figs. 4-2, 4-3). Trends in step length variability and stride time variability were not affected when stride times and step lengths were scaled to anthropometrics and speed (Hof 1996).

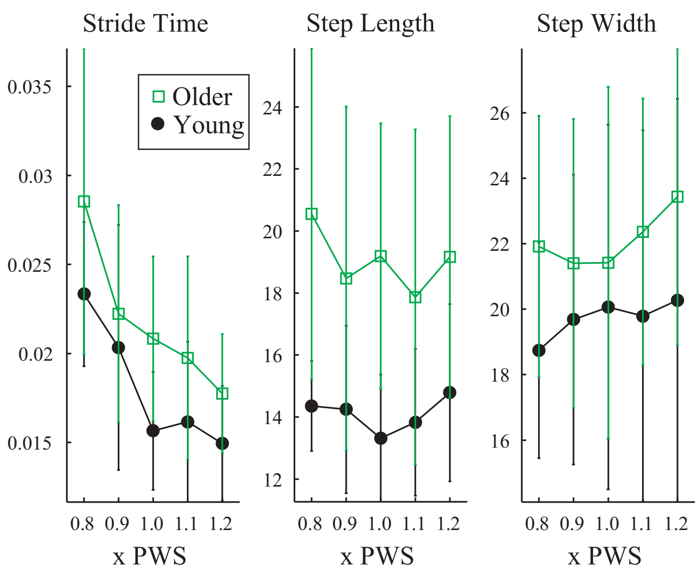


Figure 4-2. Spatio-temporal Variability vs. Speed.

Stride time ($p < 0.02$) and step length ($p < 0.005$) trended toward more variablity in older adults. Stride time ($p < 0.001$) was also affected by walking speed. Error bars denote standard deviation.

Walking speed significantly affected the variability of stride time, hip abduction/adduction angle, knee varus/valgus angle, knee internal/external rotation, and all trunk motions ($p < 0.002$; Figs. 4-2,3,4). A significant interaction between age and speed was found only for trunk roll MeanSD ($p < 0.002$). Age-effects for stride time variability ($p = 0.252$), step length variability ($p = 0.618$), MeanSD of trunk pitch ($p = 0.802$) and roll ($p = 0.390$) were not significant when the composite Strength and ROM were included as covariates. The relationships of these 4 measures to the covariates are shown in Figure 4-5.

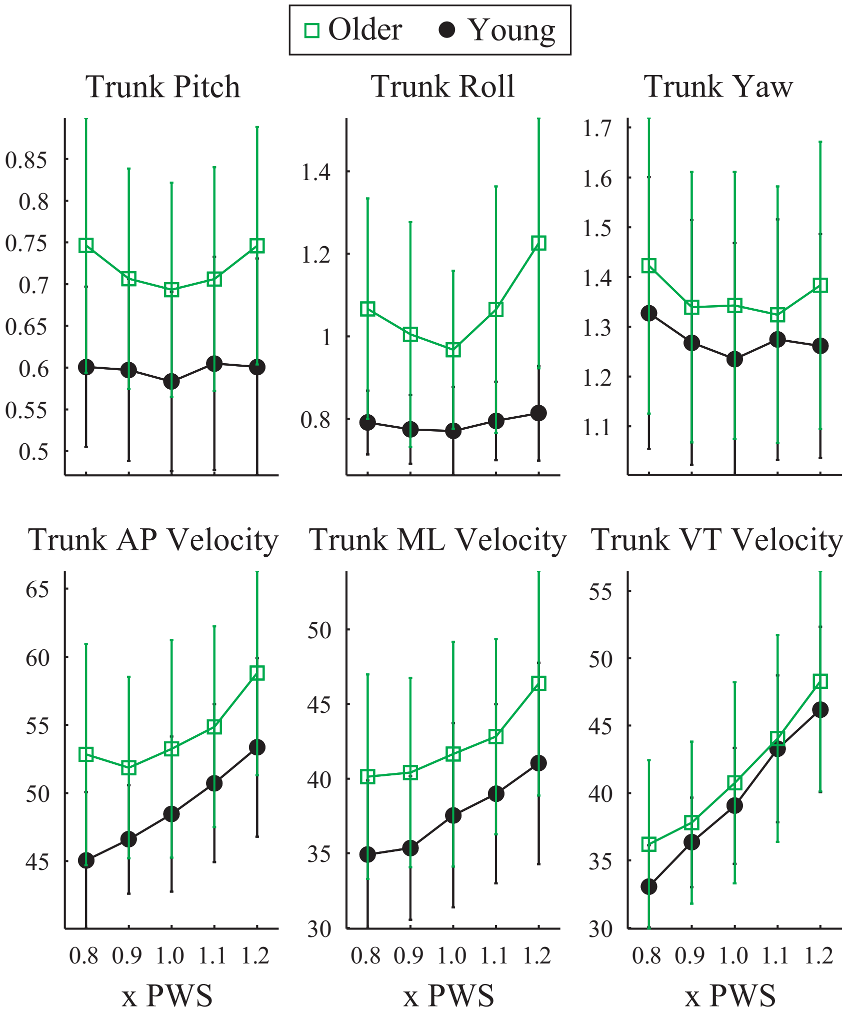


Figure 4-3. MeanSD of Trunk Motion vs. Speed.

Variability of trunk roll angle ($p < 0.001$) angles was larger in older adults. Speed effects were significant for all measures ($p < 0.002$). Interaction effects were significant ($p < 0.001$) only for trunk roll MeanSD. Error bars denote standard deviation.

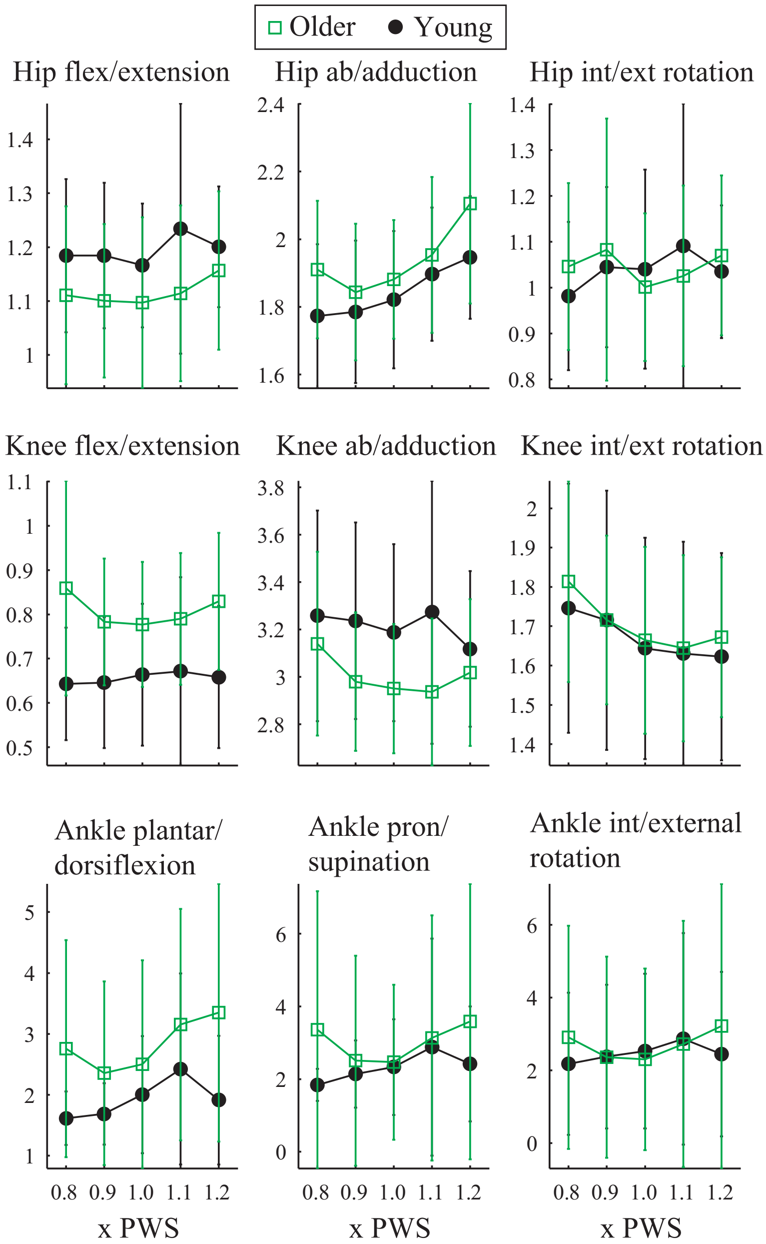


Figure 4-4. MeanSD of 3D Joint Angles vs. Speed.

Speed effects were significant in MeanSD of hip abduction/adduction angle, knee internal/external rotation, and knee abduction/adduction ($p < 0.002$). Young adults exhibited higher MeanSD of hip flexion/extension angle and knee abduction/adduction angles, but the differences were not significant ($p > 0.05$). Error bars denote standard deviation.

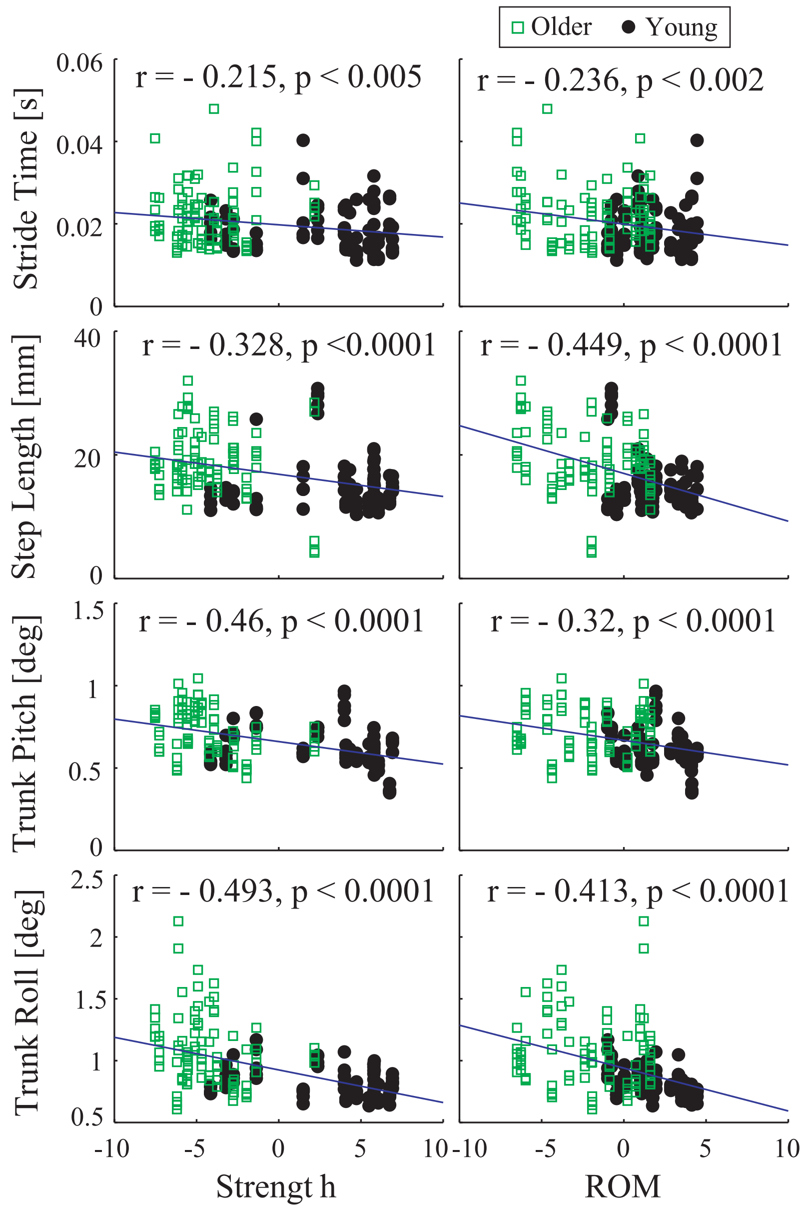


Figure 4-5. Relationship of Age-Sensitive Variability Measures and Covariates.

Significant age-related differences in variability could be explained by either strength or ROM as covariates.

Table 4. P-values for Variability Comparisons vs. Speed and Age

Variable	Age	Speed	Age x Speed	Age, adj by strength	Age, adj by ROM	Age, adj by both
Trunk Pitch	0.022	0.001	0.261	0.913	0.186	0.802
Trunk Roll	0.0003	0.001	0.002	0.138	0.020	0.390
Trunk Yaw	0.593	<0.001	0.600			
AP Vel.	0.124	<0.001	0.047			
ML Vel.	0.134	<0.001	0.713			
VT Vel.	0.638	<0.001	0.130			
Stride Time	0.018	<0.001	0.259	0.112	0.138	0.252
Step Length	0.005	0.170	0.361	0.115	0.032	0.618
Step width	0.151	0.162	0.568			
Hip flex/ext.	0.099	0.056	0.555			
Hip ab/add.	0.372	<0.001	0.042			
Hip rotation	0.577	0.550	0.394			
Knee flex.	0.051	0.374	0.109			
Knee ab/ad	0.062	0.001	0.025			
Knee rot.	0.982	0.001	0.481			
Ankle plant.	0.839	0.409	0.635			
Ankle pron.	0.521	0.767	0.479			
Ankle rot.	0.786	0.956	0.484			

DISCUSSION

Increased gait variability is a risk factor for falls in older adults (Lord, Lloyd et al. 1996; Hausdorff, Rios et al. 2001). However, because older adults typically walk slower, and gait variability varies with walking speed, it is difficult to establish whether the increased variability observed in older adults comes from slower walking or other causes. By separating possible confounding effects of walking speed, we can better identify risk factors for falls. Greater variability existed in older adults for stride time, step length, and trunk roll independent of differences in speed. Furthermore, these differences in variability were explained by lower leg strength and range of motion. Other variability measures were influenced mainly by walking speed instead. A significant Age x Speed interaction was present only in trunk roll. Thus, variability in older adults was not affected by changes in walking speed more than young adults.

Previous studies (Öberg, Karsznia et al. 1993; Öberg, Karsznia et al. 1994) have described group differences using self-selected speeds, making direct comparisons between groups and walking speeds difficult. By directly controlling walking speeds, we avoided this problem. Also, because gait variability is speed-sensitive, testing everyone at the same speed is confounded by each person's PWS and their own sensitivity to walking speed (Moe-Nilssen and Helbostad 2005). Here, all speeds were relative to PWS, which was not significantly different between the groups. This allowed a direct group comparison. Age-related changes in variability were found independent from the influence of walking speeds, demonstrating for the first time that age-related factors other than speed contribute to the increased gait variability. While some have suggested that this increase may be due to underlying pathologies rather than normal aging (Hausdorff, Rios et al. 2001), our results indicate that changes in gait variability exist in healthy normal aging.

This increased age-related variability was very noticeable in trunk roll angle. This is supported by the literature, where the roll motion is not passively stable in a walking model (Kuo 1999), and fall risk was predicted by step width variability (Hausdorff, Rios et al. 2001). We did not find a significant age-effect ($p < 0.16$) for step width variability, although a previous study did (Owings and Grabiner 2004). This discrepancy exists perhaps because the older adults were quite healthy, and could complete the entire protocol including ~1 hour total of walking. Nevertheless, age-related differences were found in other variables. Such differences may have come from treadmill experience, but there is no reason to believe that young subjects were more experienced, especially when the older subjects were healthy and active.

Significant speed-sensitivity was seen in many variables, especially trunk motion, despite the relatively narrow range of speeds investigated. Variability-speed relationships were not all U-shaped as previously demonstrated, but this may be due to the narrower speed range. Speed effects on variability are more pronounced at very high or very low speeds (±40% PWS) (Dingwell and Marin 2006), but most older adults in this study could not walk any faster comfortably. The speed range used here (±20% PWS) reflects the literature, where older adults tend to walk up to 20% slower than young adults (Prince, Corriveau et al. 1997).

One potential limitation of this study was that subjects walked on a motorized treadmill. Treadmills may artificially reduce the natural variability, compared to overground walking, because walking speed is strictly enforced (Dingwell, Cusumano et al. 2001; Wass, Taylor et al. 2005). Psychophysical differences, such as the lack of optic flow, may also affect variability. However, all subjects were tested under the same experimental conditions and relative to their own walking speeds. Therefore, overground walking may yield slightly larger values for the measures quantified. However, the observed differences between groups and speeds are expected to remain. Having more subjects may have yielded more significant comparisons, but there was still sufficient statistical power to find significant differences in these variables, even after Bonferroni correction.

If slower walking speeds do not explain the age-related changes in gait variability, then what does? Using strength and/or ROM as covariates eliminated the observed age-effects (Figure 4-5), suggesting that decreased strength and flexibility explain the increased gait variability in older adults, and thus may be linked to fall risk. Knee extension strength was correlated with stride time variability in a prospective falls study (Hausdorff, Rios et al. 2001). Strength training and stretching interventions have reduced fall risk in some older adults (Barnett, Smith et al. 2003; Ballard, McFarland et al. 2004; Liu-Ambrose, Khan et al. 2004; Liu-Ambrose, Khan et al. 2004; Faber, Bosscher et al. 2006). Since gait variability also predicts fall risk, this suggests that gait variability should be also modified by these interventions. However, strength interventions only minimally affect gait variability (Hausdorff, Nelson et al. 2001) or body COM excursion (Krebs, Jette et al. 1998), which raises a question about the validity of gait variability as a predictor of falls. This discrepancy may be because these interventions seem to help only the healthy "pre-frail" older adults (Faber, Bosscher et al. 2006), suggesting that the relationship between fall risk, gait variability, and strength may be different in healthy and "frail" populations. In future work, separating the effects of walking speed may help clarify the picture in older adults who fall or are "frail."

Although trunk roll variability was correlated to leg strength and flexibility, it is not immediately obvious why. Strength and flexibility were measured only in the sagittal plane, while trunk roll measures frontal plane motion. Although these covariates explain the group differences in a statistical sense, their relationship to trunk roll variability is still weak (Figure 4-5). This may be because trunk roll variability may be related to the strength of hip adductor/abductors, and the oblique muscles which were not measured in this study. Increased trunk roll variability also may be caused by other factors associated with aging, such as decline in nervous system function. The relationship of age-related difference in trunk roll variability and muscles involved in frontal plane motion needs to be investigated in the future. The change in motor control strategies due to age, specifically how it may affect trunk roll during gait, also needs to be investigated.

Chapter 5: Does Slower Walking in the Elderly Improve Gait Stability?

INTRODUCTION

To understand falls in older adults that occur during walking, it is important to understand how aging affects gait and gait stability. Slower speed is observed in healthy older adults, and is more pronounced in those with a variety of clinical conditions (Alexander 1996). Slow gait speed have predicted prospective fall risk in some studies (Luukinen, Koski et al. 1995; Bergland, Jarnlo et al. 2003), but not in others (Maki 1997; Menz, Lord et al. 2003). Thus it is not clear if slower walking can be used to predict fall risk. Also, to use walking speed as a useful diagnostic tool, the various causes of slower walking have to be clarified. However, it is not entirely clear why older adults, especially otherwise healthy ones, walk slower.

Some have suggested that slower walking speeds stem from physical constraints that prevent faster walking, such as decreased strength (Olney, Griffin et al. 1994; Burnfield, Josephson et al. 2000; DeVita and Hortobagyi 2000; Bohannon 2001). Strength training increased walking speed in older adults (Chandler, Duncan et al. 1998; Holviala, Sallinen et al. 2006). However, increasing walking speed by strength training did not improve fall risk (Chandler, Duncan et al. 1998), and in fact might instead put older adults at greater risk of falls (Pavol, Owings et al. 1999; Pavol, Owings et al. 2001). Thus strength is a factor in slower walking this is not the complete picture.

Others have suggested that slower walking may be a pro-active strategy to improve stability, by contributing to feeling "safer" (Winter, Patla et al. 1990; Gardner and Montgomery 2001; Menz, Lord et al. 2003). Gait speed was related to fear of falls, not fall risk, in older adults (Maki 1997), suggesting that older adults slow down as a precautionary measure to be more stable. Local dynamic instability decreases with slower walking speeds in young adults, confirming that slowing down does improve gait stability, despite increases in variability (Dingwell and Marin 2006) However, this finding has not been demonstrated in older adults.

To understand better the risk factors for falls associated with slower gait speed, we tested whether gait speed is associated with improved gait stability, leg strength, and range of motion in healthy older adults as in young adults. Dynamic stability was quantified at multiple controlled walking speeds to allow better comparisons (Kang and Dingwell 2007). Leg strength and range of motion were also tested as potential mediating factors of gait stability.

METHODS

Eighteen healthy older adults (age 65-85) and 17 height-, weight-, gender-matched healthy young adults (age 18-28), participated with informed consent as described in Chapter 3. Kinematics, strengths and ROM were measured, but for this study, only the trunk motion was studied as a function of age and the five walking speeds. Speeds presented were 80, 90, 100, 110, and 120% of each individual's preferred walking speed. Trunk motion was studied because it accounts for over half of the body mass (Winter 1990), and the control of the trunk segment affects gait dynamics greatly (Winter, MacKinnon et al. 1993). If the trunk stability is not maintained, then a fall may ensue.

Data Processing

The trunk segment was modeled as a rigid body with six degrees-of-freedom. The motion of the trunk segment was described using linear and angular velocities $[\dot{x}\ \dot{y}\ \dot{z}\ \dot{\theta}\ \dot{\phi}\ \dot{\psi}]$ and accelerations $[\ddot{x}\ \ddot{y}\ \ddot{z}\ \ddot{\theta}\ \ddot{\phi}\ \ddot{\psi}]$, forming a 12-dimensional state space (Eqn 5-1).

$$S(t) = [\dot{x}, \dot{y}, \dot{z}, \ddot{x}, \ddot{y}, \ddot{z}, \dot{\theta}, \dot{\phi}, \dot{\psi}, \ddot{\theta}, \ddot{\phi}, \ddot{\psi}] \qquad (5\text{-}1)$$

The linear motions of the trunk were defined from the linear excursions of a virtual center marker, defined as the average location of the six torso markers. 3D trunk rotations were defined using the Cardan Y-x'-z'' (tilt-obliquity-rotation) convention relative to the laboratory reference frame. Velocities and accelerations were calculated using the standard 3-point difference formula. Trunk velocities and accelerations were used instead of absolute trunk positions to reduce the non-stationarity in the data (Dingwell and Marin 2006). The non-stationarity in position variable comes from the subject as they drift around on the treadmill while they walk. These state variables were then low-pass filtered with a cutoff of 10 Hz using a Butterworth filter. This minimized the effects of measurement noise and non-rigid behavior (subtle bending, raising the shoulders, twisting, etc.) of the trunk. Mean log divergence to quantify local dynamic stability and Floquet multiplers (FM) to quantify orbital stability were calculated at each speed, as described in Chapter 3. FM did not vary noticeably during the gait cycle as previously found (Dingwell and Kang 2007). Therefore, FM results are reported from 0, 25, 50, and 75% of the gait cycle.

Statistics

Exponents λ^*_S, λ^*_L and FM at 0, 25, 50, and 75% of the gait cycle were compared between age groups and speeds using a general linear model mixed-model ANOVA using

SPSS 14 (SPSS, Chicago IL). The analysis was repeated as an ANCOVA, where the composite strength and ROM scores were included as covariates.

RESULTS

The PWS of older adults were no different from young adults (all subjects: 1.29 ± 0.13 m/s, $p = 0.86$; Table 2). Older adults had lower composite strength and ROM scores ($p < 0.0001$; see Table 2 and Appendix C).

Mean Log Divergence curves are shown in Figure 3. λ^*_S was larger in older adults ($p < 10^{-13}$). This indicated that older adults exhibited higher sensitivity to perturbations. λ^*_S increased with speed ($p < 0.001$), and this effect was a bit more prounced in older adults (interaction $p = 0.007$). λ^*_L did not vary with age ($p = 0.192$), but it increased with speed ($p < 0.001$; Fig. X). Thus slower walking speeds were less sensitive to perturbations. These speed effects were similar to previous findings (Dingwell and Marin 2006; England and Granata 2006).

Age effects were less, but remained significant, when either Strength ($p < 10^{-8}$) or ROM ($p < 10^{-9}$) composite score was included as covariate(s) ($p < 10^{-6}$ with both; Fig. 5). Strength and range of motion may help explain some of age-related differences in local dynamic stability, but other age-related factors may explain this difference better.

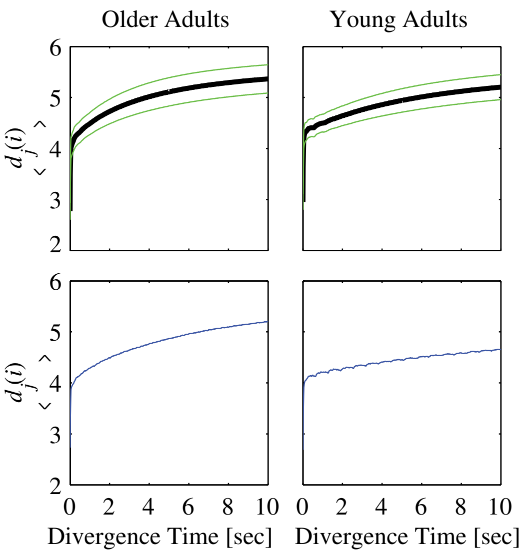


Figure 5-1. Sample local dynamic stability

Divergence curves are displayed for the 1x PWS speed. Top: Group averages with standard deviation bars (dotted line). Bottom: A typical trial from each group (left: older adult, right: young adult). Older adults displayed greater between-subjects variability compared to young adults, and larger divergence values, even though their walking speeds were not different.

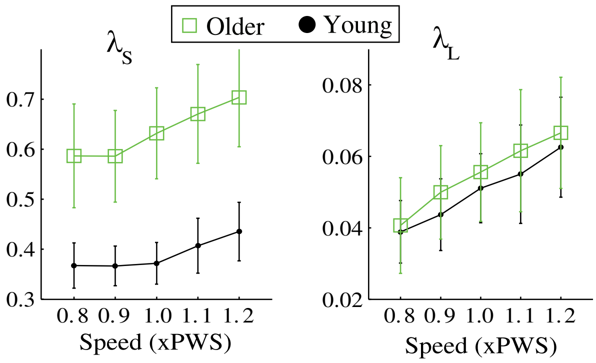


Figure 5-2. Local stability vs. Speed and Age

Error bars denote standard deviations within each group.

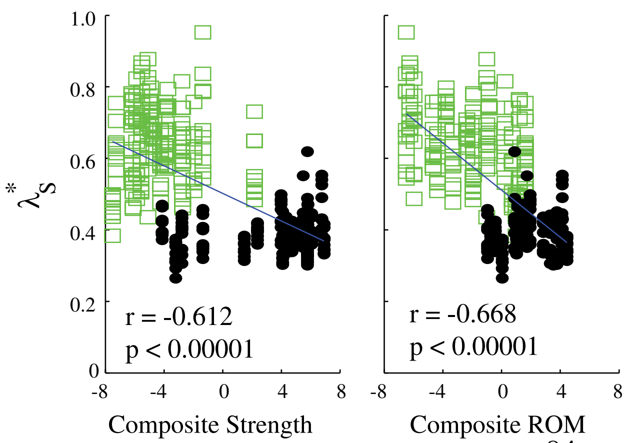


Figure 5-3. Local stability vs. Covariates

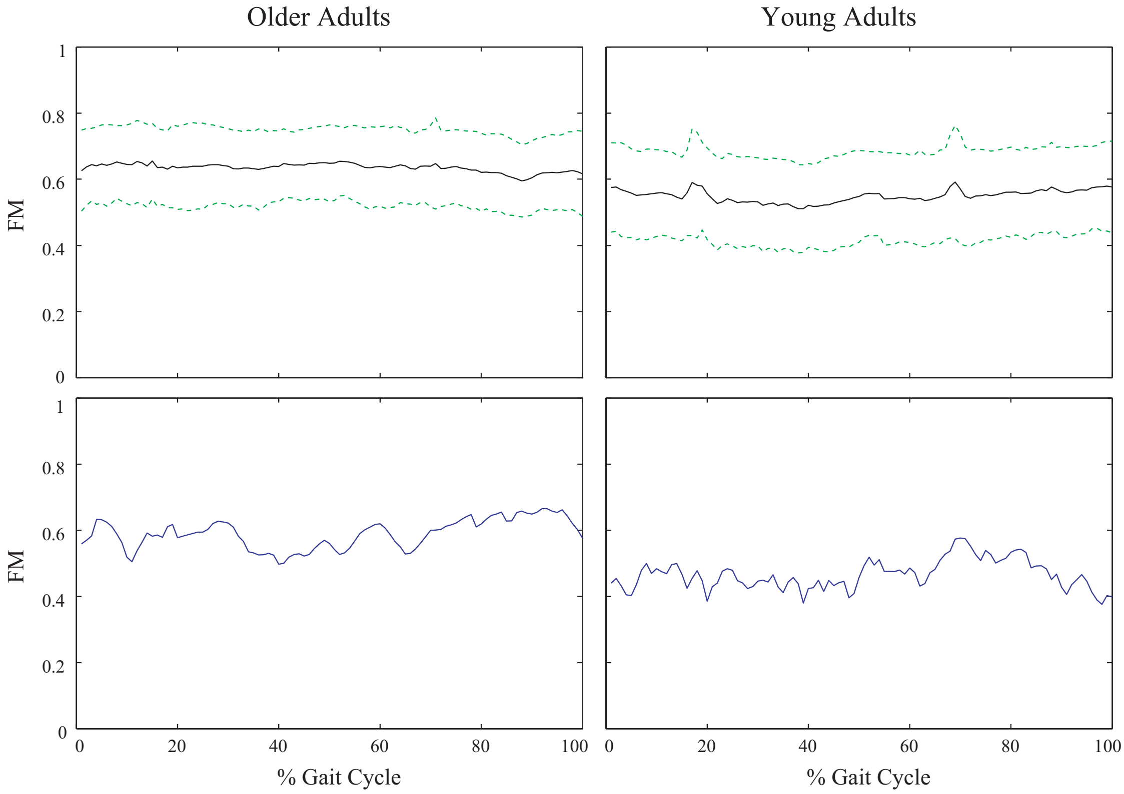


Figure 5-4. Floquet Multipliers over the Gait Cycle.

FM values are displayed for the 1x PWS speed. Top: Group averages with standard deviation bars (dotted line). Bottom: A typical trial from each group (left: older adult, right: young adult). FM was consistent across the gait cycle. Older adults displayed larger FM values. A few trials displayed a spike at 20% or 70% of the gait cycle, but it was not consistent between subjects or groups.

Largest magnitude Floquet multipliers (FM) were almost constant during the gait cycle, and did not vary in any consistent manner over the gait cycle (Figure 5-4). Older adults exhibited higher FM values at 0, 25, and 75% of the gait cycle ($p < 0.001$), and trended toward the same at 50% ($p < 0.007$). FM were larger at higher speeds at 75% ($p < 0.001$), where FM at 80, 90% were less than 110, 120% ($p < 0.0027$, pairwise comparisons), but only trended toward significance at other parts of the gait cycle ($p < 0.02$), and not significant at 0% ($p < 0.4$). When strength scores were included as a covariate, age-effects remained significant ($p < 0.005$) only at 75% of the gait cycle. When ROM was included, all age effects were not significant ($p = 0.010, 0.033, 0.11, 0.007$ for the 0, 25, 50 and 75% of the cycle).

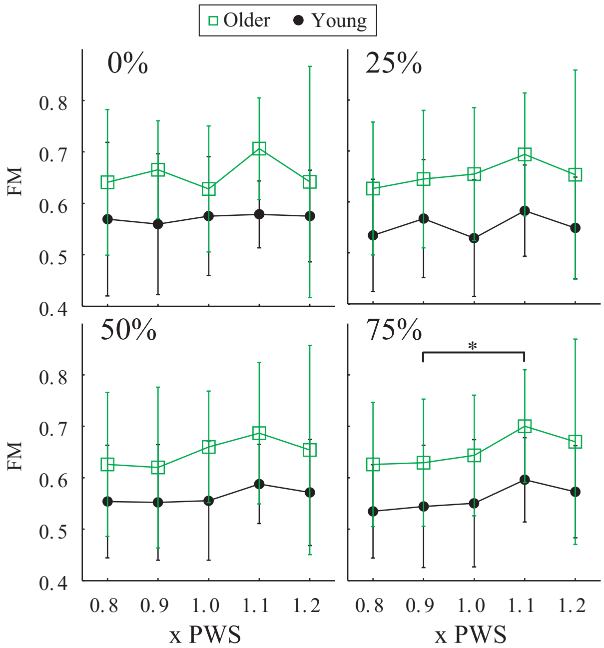


Figure 5-5. Floquet Multipliers vs. Speed.

FM values are shown at 0, 25%, 50% and 75 of the gait cycle. Older adults (squares) displayed higher FM. At 75% (bottom right), FM at larger speeds were larger than that at slower speeds, while the speed-effect was less noticeable at other parts of the gait cycle. Error bars denote standard deviations within each group. the horizontal bracket denotes significant Tukey's LSD post-hoc comparisons at $p < 0.005$. At 75% of the gait cycle, FM at 0.8x and 0.9x speeds were significantly different from those at both 1.1x and 1.2x speeds.

Table 5. P-values for Stability Measures

	λ^*_S	λ^*_L	0%	25%	50%	75%
Age	<0.0001	0.192	0.0052	0.0013	0.0066	0.0024
Speed	<0.0001	<0.0001	0.344	0.0172	0.0137	<0.0001
Age x Speed	0.0068	0.577	0.428	0.217	0.3569	0.525
Age adj by strength	<0.0001	N/A*	0.0100	0.0110	0.0310	0.0035
Age adj by ROM	<0.0001	N/A*	0.0112	0.0196	0.0920	0.0114
Age adj by both	<0.0001	N/A*	0.0107	0.0330	0.1101	0.0068

* Main-effect for age was not significant, therefore not applicable

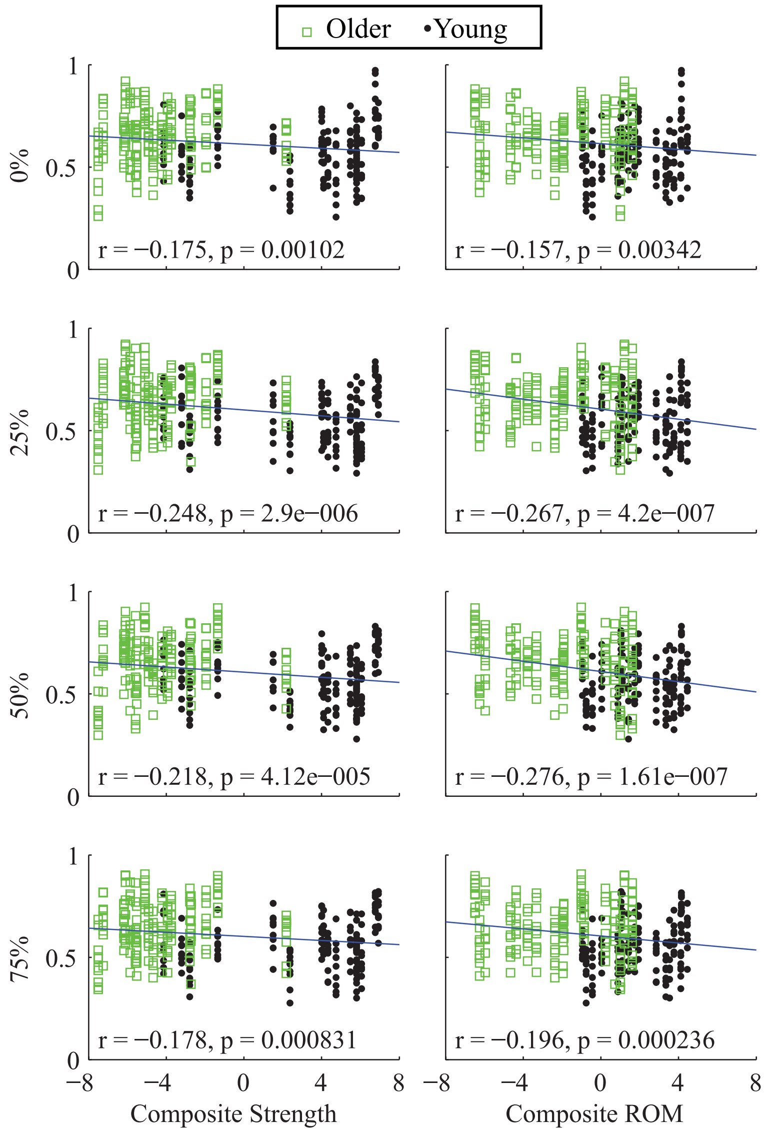


Figure 5-6. Relationship of FM values to Strength and ROM

DISCUSSION

To understand falls in the elderly better, we need to understand how gait dynamics and stability are affected by the aging. The cause of slower walking, commonly observed in older adults, whether chosen as a strategy, or necessitated by physiological deterioration, is still debated. We addressed this issue by systematically comparing stability at different walking speeds in healthy young and older adults.

Slower speeds were associated with a lower λ^*S in older adults, reflecting less sensitivity to perturbations when walking slower. This is similar to previous findings in young adults (Dingwell and Marin 2006; England and Granata 2006). As one would expect, faster walking is more susceptible to deviations away from the original or intended kinematic trajectory due a perturbation. Also, slower walking speeds were orbitally stable in both young and older adults, although(Kang and Dingwell 2006) the effect of walking speed was small. Higher FM at faster speeds demonstrated that it is more difficult to recover from perturbations at these faster speeds. In previous studies, speed did not affect orbital stability in young adults (Dingwell, Kang et al. 2007). Speed effects, while significant, were small in this current study as well.

Higher λ^* values in older adults indicate that their gait was more sensitive to perturbations. If given a perturbation, their gait would deviate from its original trajectory faster than in young adults. Thus older adults were less stable and this difference was present even when speed effects were separated. Larger Floquet multiplier magnitudes (FM) in older adults suggest that their walking is less stable than young adults, independent of walking speed. These age-differences in FM could be explained with strength and ROM as covariates, suggesting that the difference in stability is mediated by these age-related changes in strength and flexibility.

The age-related differences and the small speed-related differences in FM suggest that orbital stability reflects the inherent capacity to deal with perturbations during gait. FM did not vary much within the young or older adult groups between walking speeds, as the inherent capacity to recover from perturbations within each person would not vary depending on the task. FM were significantly different between the age groups, which may reflect the decreased capacity to recover from perturbations in older adults. These findings together suggest that slower walking in healthy older adults may be chosen. Older adults seem to be more susceptible to perturbations, yet their capacity to respond diminishes. In response, they may choose to walk slower to reduce the effect of perturbations given their ability to respond.

Some previous studies have suggested that physical limiting factors such as muscle weakness or loss of flexibility cause slower walking in older adults. Others have suggested older adults choose to walk slower for their sense of safety. Our results support this second view, as older adults also experience better orbital stability at slower walking speeds. In our subjects, preferred walking speed was not related to the strength or ROM scores, and thus the older adults did not walk any slower than young adults because of their deficits in strength or flexibility. These older adults exhibited isometric leg strengths similar to the community-dwelling elderly in the literature (Melzer, Benjuya et al. 2004), except for stronger knee flexors (75.1±23.2 vs. 48.9±6.7 Nm). However, more decrease in strength was associated with slower walking speed (Chandler, Duncan et al. 1998). Therefore, it seems that up to a certain amount of strength decline, older adults may not be forced to walk slower, even though they may choose to do so for stability reasons.

This reason may be not apply to a clinical population, with cardiovascular, metabolic, nervous, and other considerations that would limit the ability to walk faster.

These populations would also choose to walk slower for different reasons. However, these would be unlikely reasons for healthy older adults. Although the healthy elderly studied here did not walk slower because of deficits in strength or flexibility, these deficits did explain the decreased orbital stability in these older adults. Since strength and flexibility deficits explain the age-related differences in stability, we need to study whether interventions that improve strength and flexibility also improve gait stability.

Statistical measures such as variability can be used to predict falls, but they do not tell us why falls occur, or quantify how the locomotor system responds to perturbations that may cause a stumble or a fall (Dingwell and Cusumano 2000). Thus, they do not help us understand how stability is maintained during walking. More appropriate measures that quantify actual walking stability were used in this study. The relationships between stability and speed in the trunk are different from that of variability and speed as shown in (Kang and Dingwell 2007).

One potential limitation of this study was that subjects walked on a motorized treadmill. Treadmills may artificially reduce the natural variability and local instability (Dingwell, Cusumano et al. 2001; Wass, Taylor et al. 2005), and may not reflect overground walking in older adults (Wass, Taylor et al. 2005), because walking speed is strictly enforced on the treadmill. However, there were only minimal differences in orbital stability between treadmill and overground walking in young adults (Dingwell and Kang 2007). Also, all subjects were tested at the same experimental conditions and relative to their own walking speeds, which were not different between groups. Therefore, repeating this study overground may yield slightly different dependent values. However, the observed differences between groups and speeds are expected to remain.

In summary, we found that older adults walk with less stability, that slower walking speed improves stability, and the age-related differences in stability were explained partly by decline in strength and flexibility. Deficits in strength or flexibility in themselves do not seem to cause slower walking in healthy elderly. Further work is needed to see if improving strength or flexibility would improve gait stability in the elderly. Also, the relationship of age-related changes in gait stability to cognitive and nervous function remains to be explored.

Chapter 6: Segment Height and Stability during Gait

INTRODUCTION

In order to understand falls in older adults that occur during walking, it is important to understand how movement is controlled during walking, and how stability is maintained by the nervous system. The trunk segment consists of over half of body mass (Winter 1990), and greatly influences the dynamics of the rest of the body. Not simply a mass to be transported by the legs, the trunk performs functions related to support and stabilization during walking (Winter, MacKinnon et al. 1993; Prince, Winter et al. 1994; Kavanagh, Morrison et al. 2006). Active control of the trunk motion is believed to enable stability to be maintained during walking (Winter, MacKinnon et al. 1993; Prince, Winter et al. 1994). Because of its mass, if the movement of the trunk segment is not controlled well, then a fall is likely to occur.

Research suggests that control of superior segments takes precedence over inferior segments of the body during walking. Shocks from walking are absorbed as they move up the kinematic chain (inferior to superior segments) toward the head (Ratcliffe and Holt 1997). By the time they reaches to the head, acceleration amplitudes dampen to ~20% of that measured at the shank level (Kavanagh, Morrison et al. 2006). This suggests that after prioritizing the control of trunk movement, the nervous system would stabilize the pelvis next, and thigh the next, and so on, such that the feet are least stable. This would occur as the inferior segments adjust their motion to stabilize the trunk segment. Thus, the stability of body segments would also follow the order of the kinematic chain or segment height, where superior segments are more stable than inferior segments.

There is some evidence that older men walk with less trunk acceleration amplitudes than young adults after adjusting for walking speed (Kavanagh, Barrett et al. 2004). However, it is not clear how aging or other clinical conditions affect the control of the trunk segment relative to lower segments. Floquet multipliers (FM) based on the vector from the heel to the center of mass at each step were larger in older adults with a history of falls compared to healthy older adults, and FM was even less in young adults (Granata and Lockhart 2006). Although this study tells us the importance of the relationship of trunk and the feet, it only provides a single measure of stability for the entire body, and did not tell us the relationship between individual segments themselves. Therefore, in this study, we tested if superior segments were more stable than inferior segments in healthy older adults compared to young adults, and whether the stability relationships among segments were altered with age.

METHODS

Eighteen healthy older adults (age 65-85) and 17 height-, weight-, gender-matched healthy young adults (age 18-28), participated with informed consent as approved by the University of Texas Institutional Review Board (Table 2). Kinematics, strengths and range of motion data were collected as described in Chapter 3. Only the preferred walking speed was considered for this chapter.

State Space Description of Segments

For each segment, the trunk, pelvis, left thigh, left shank, and the left foot, a 12-dimensional state-space was defined. For each segment, the mean location of the markers was used as a basis for position data. Segment angles relative to global coordinate system were calculated using tilt-obliquity-rotation sequence as above. From these values, the linear and angular velocities were calculated using the 3-point difference formula as described in Chapter 5.

The motion of the each segment and its response to these perturbations was described using linear and angular velocities $[\dot{x}\ \dot{y}\ \dot{z}\ \dot{\theta}\ \dot{\phi}\ \dot{\psi}]$ and their time-delays, forming a 12-dimensional state space (Eqn 6-1). A state-space based on velocities and accelerations was not used as in Aim 2 because of the disparity in the relative magnitudes of the state variables. The inferior segments exhibit larger motions, and therefore their accelerations can be very high compared to superior segments, as well as the size disparity between the state variables. Because the foot motion is much larger than the trunk motion, the linear acceleration values were a few orders of magnitude larger than other variables. This skewed the state-space so much that spurious results were found. To resolve this problem, the analysis was performed with a reconstructed state space consisting of the velocity variables and their time-delayed copies (Packard, Crutchfield et al. 1980).

$$\begin{aligned} S(t) = [&\dot{x}(t) \quad \dot{y}(t) \quad \dot{z}(t) \quad \dot{\theta}(t) \quad \dot{\phi}(t) \quad \dot{\psi}(t) \ldots \\ &\dot{x}(t-\tau_1) \quad \dot{y}(t-\tau_2) \quad \dot{z}(t-\tau_3) \quad \dot{\theta}(t-\tau_4) \quad \dot{\phi}(t-\tau_5) \quad \dot{\psi}(t-\tau_6)] \end{aligned} \tag{6-1}$$

Time delays were calculated from the first minimum of the average mutual information function of each time series (Fraser and Swinney 1986).

Mean log divergence and Floquet multipliers were calculated (Dingwell and Kang 2007) for each segment as described previously in Chapter 5. Mean log divergence curves and the magnitudes of the largest Floquet multipliers (FM) were quantified from the state-space defined above, using methods as described previously in Chapter 5. FM did not vary noticeably during the gait cycle (Dingwell and Kang 2007). Therefore, FM results are reported from 0, 25, 50, and 75% of the gait cycle.

Variability of the kinematics was quantified in two different ways. RMS distance of the points on the Poincaré section from the fixed point was calculated at each % of the gait cycle (see Appendix E for more details). Then the RMS values were averaged across the gait cycle, to arrive at MeanRMS, similar to MeanSD Chapter 4. MeanRMS values were also normalized to the size of the state-space by the RMS radius of the data points from the centroid of the data points (Figure 6-1). Commonly, variability of a gait measure is calculated by time-normalizing, then measuring the variability. However, this procedure can distort the results, as the dynamics may change between strides. Also, these variability measures only describe one variable at a time. To address these limitations of the traditional approach, we described the variability of each segment motion in state-space. Rather than considering one state-variable at a particular point of the gait cycle, we consider all state variables on the Poincaré section at a particular point of the gait cycle. Variability is described by the spread of the trajectories on the Poincaré section (Fig 6-1). This gave us an aggregate measure of variability of the system that considers the dynamics of all state variables.

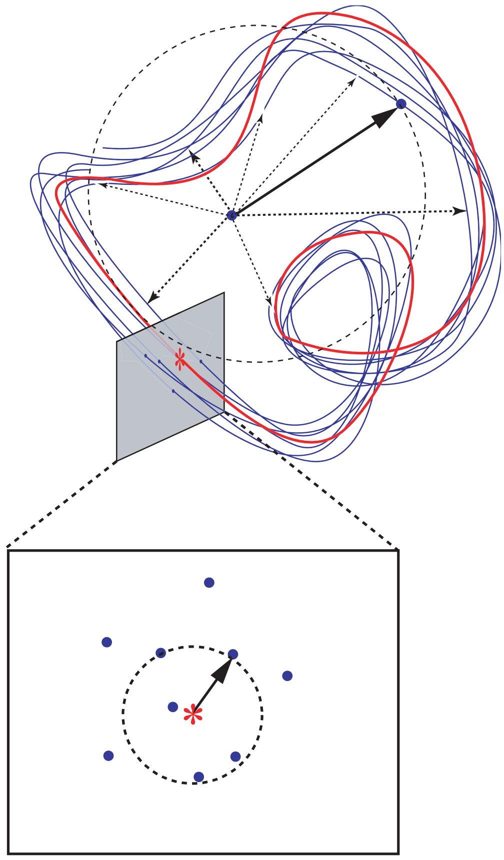


Figure 6-1. Calculation of Variability in State-space.

Illustration of a Poincaré section in state space. RMS distance (dotted black circle) of individual strides (blue dots) from the mean (star) was defined on the Poincaré section was calculated (bottom). Mean of the RMS distances over the gait cycle (MeanRMS) was used to define the variability metric. The RMS distance (RMSD) from the centroid of the data points to each data point (dotted circle) was used to normalize the MeanRMS (top).

Statistics

Mean log divergence slopes λ^*_S, λ^*_L, FM at 0, 25, 50, and 75% of the gait cycle, MeanSD and MeanSD/RMSD were compared between age groups and segments using a repeated-measures ANOVA using SPSS 14 (SPSS, Chicago IL). The divergence fit terms were log-transformed to normalize the distribution (Berry 1987). The analysis was repeated as an ANCOVA, where the composite strength and ROM scores were included as covariates. To reduce type II error, a Bonferroni correction of $\alpha = 0.05 \div 11 \approx 0.0045$ was used.

RESULTS

Mean Log Divergence curves are shown in Figure X. λ^*_S was larger in older adults ($p < 0.0001$). This indicated that older adults exhibited higher sensitivity to perturbations. λ^*_S was larger in inferior segments ($p < 0.0001$), and this effect similar in both age groups (interaction $p = 0.0162$). λ^*_L did not vary with age ($p = 0.0008$), but it increased with speed ($p < 0.0001$; Fig. 4). Thus slower walking speeds were less sensitive to perturbations. These speed effects were similar to previous findings (Dingwell and Marin 2006; England and Granata 2006).

Age effects were less, but remained significant, when either Strength ($p < 0.0003$) or ROM ($p < 0.0003$) composite score was included as covariate(s) ($p < 0.02$ with both; Fig. X). Strength and range of motion may help explain some of age-related differences in local dynamic stability, but other age-related factors may explain this difference better.

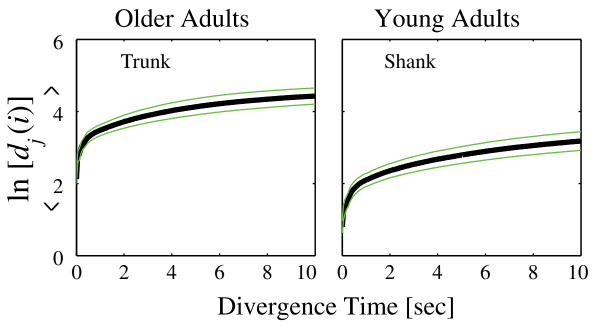


Figure 6-2. Sample Divergence Curves.

Trunk and shank segments are shown. Thick black line denotes between-subjects mean. Thin green lines denote ± 1 standard deviation.

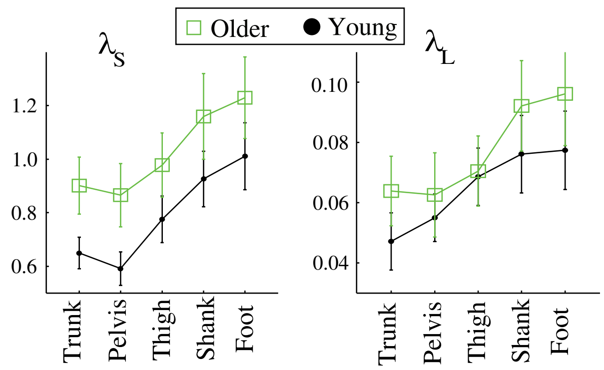


Figure 6-3. Divergence exponents vs. Segments

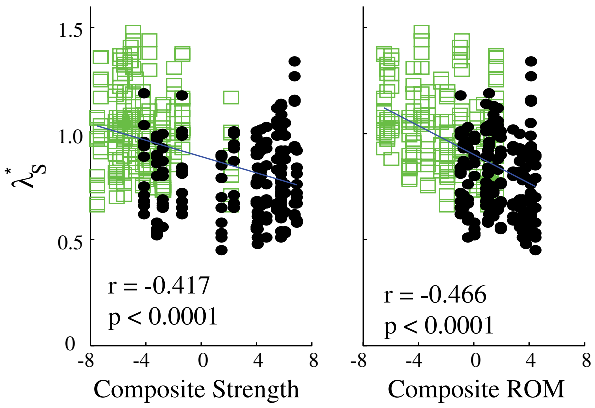


Figure 6-4. Relationships of Divergence exponents and composite strength and ROM measures.

Table 6. P-values for Stability Measures

p-values	λ^*_S	λ^*_L	0%	25%	50%	75%
Age	$<10^{-7}$	.000782	0.01285	0.01423	0.01348	0.00674
Segments	$<10^{-120}$	$<10^{-85}$	0.03538	0.00377	6.79246E-05	1.14643E-06
Age*Segs	0.0162	$<10^{-9}$	0.47834	0.34981	0.8413	0.4969
Age, adj for Str.	.0002873	0.06062	0.01806	0.02603	0.0341	0.01867
Age, adj. for ROM	.0002649	.01847	0.2003	0.2068	0.07082	0.03322
Age, adj for both	0.01171	.1498	0.101	0.12898	0.07301	0.03607

Orbital Stability

Floquet multipliers (FM) were almost constant during the gait cycle, and did not vary in any consistent manner over the gait cycle (Figure 6-5). Older adults exhibited higher FM values ($p < 0.0055$). The differences between segments were significant at all 4 points of the gait cycle ($p < 0.001$), where less orbital stability was observed in superior segments (Fig 6-6). When Strength or ROM scores were included as a covariate, age-related differences were no longer significant ($p > 0.01$; Fig 6-7).

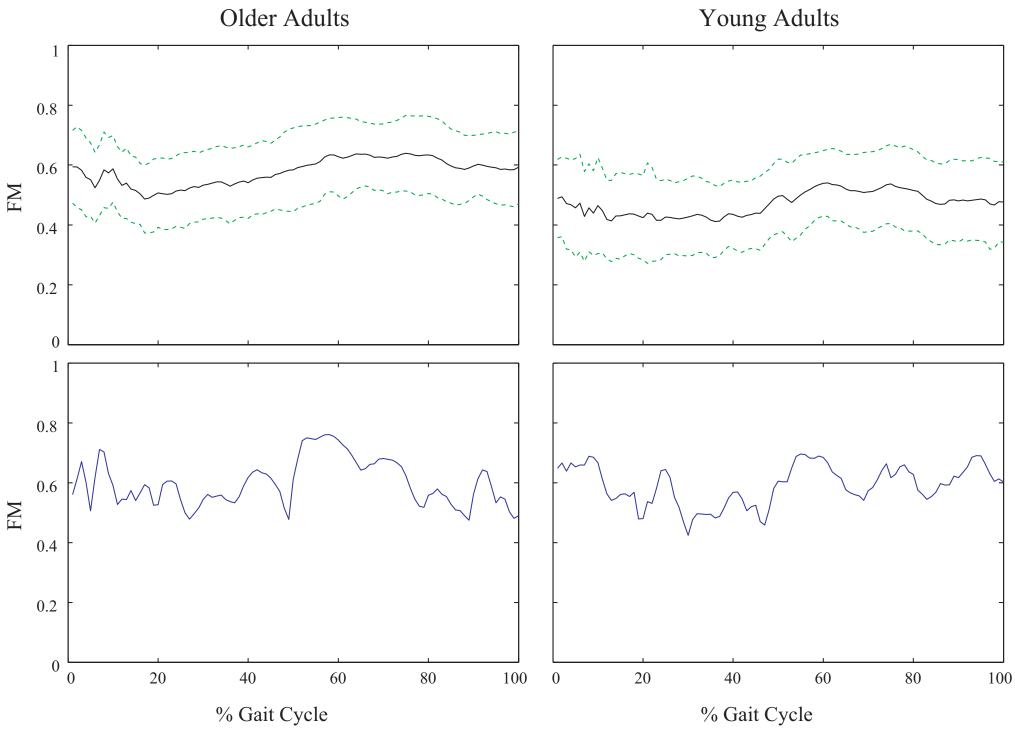


Figure 6-5. Floquet Multipliers across the Gait Cycle.

FM values are displayed for the shank segment. Top: Group averages with standard deviation bars (dotted line). Bottom: A typical trial from each group (left: older adult, right: young adult). Except for a few instances, FM lay within the unit circle, indicating orbital stability.

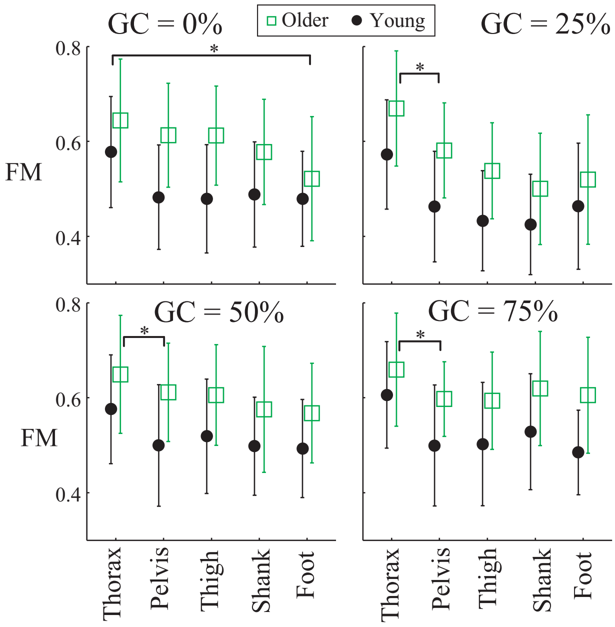


Figure 6-6. Interaction plot of Age and Segment factors for Floquet Multipliers

FM was calculated at 0%, 25% (top), 50% and 75% (bottom) of the gait cycle. Thorax displayed higher FM values than other segments. Error bars denote standard deviations within each group. Horizontal brackets denote significant Tukey's LSD post-hoc comparisons at $p < 0.005$.

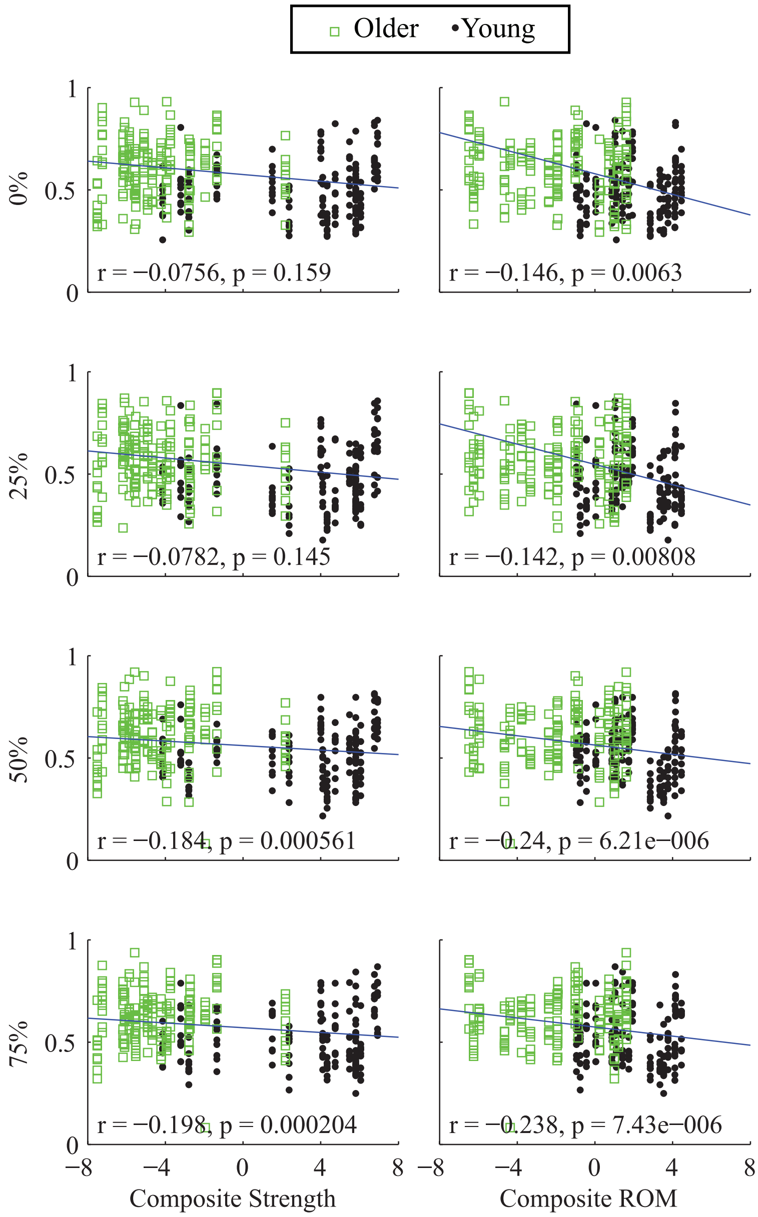


Figure 6-7. Relationship between FM with Strength and ROM

Variability

Overall variability was smaller in superior segments, as measured as MeanSD ($p < 0.001$). However, when normalized by the RMS distance (RMSD), superior segments were more variable ($p < 0.001$). Variability was not affected by age ($p > 0.1$), in contrast to orbital stability (Figure 6-8).

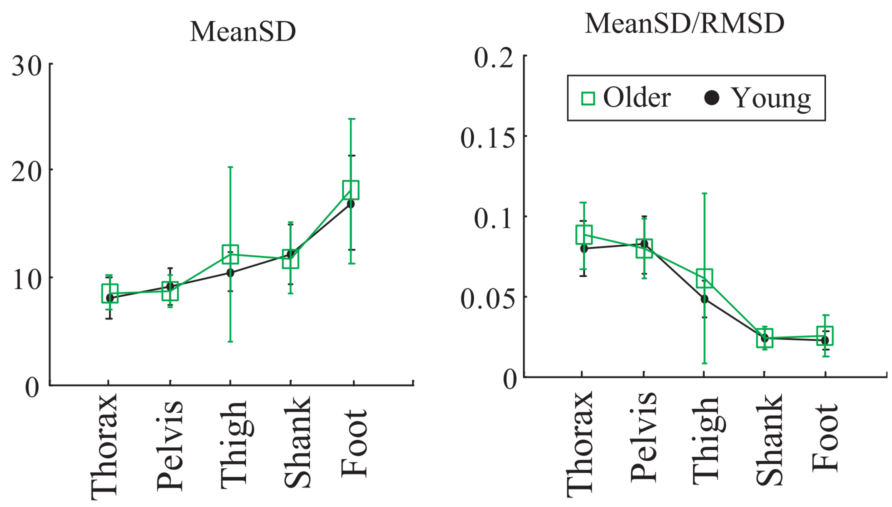


Figure 6-8. Variability of the Segment Motion in State-space.

Left: MeanSD was calculated as previously, from the RMS distance of individual strides from the mean. Error bars denote standard deviations within each group.

Overall, the results indicate that the superior segments are not any less sensitive to perturbations, and they recover at a slower rate than inferior segments.

DISCUSSION

In order to understand falls in the elderly better, we need to understand better how stability is maintained during gait, and changes that are associated with age. Active control of trunk motion is believed to enable stability to be maintained during walking (Winter, MacKinnon et al. 1993; Prince, Winter et al. 1994). Research suggests that control of superior segments takes precedence over other segments of the body during walking (Holt, Ratcliffe et al. 1999; Cromwell, Schurter et al. 2004), suggesting that stability of superior segments are prioritized over inferior segments.

Our findings suggest otherwise. The trunk segment exhibits less orbital stability than the inferior segments. Although superior segments were not necessarily less sensitive to perturbations, they do not recover as easily compared to inferior segments. This is may be because the trunk segment accounts for over 50% of the body mass, and thus takes more time to correct its motion. This finding supports the notion that the trunk segment is the key to maintaining stability, not because its stability is prioritized more, but because it is affected by perturbations for more strides.

Older adults exhibited larger FM and larger λ^*_S, λ^*_L compared to young subjects (Figures 6-3, 6-6). Thus aging does not seem to affect the motion of any single segment more than another. This may be caused the motions of the segments are linked, and therefore they affect the motion of each other. We did not find a significant age-effect in the variability measures, even though trunk roll variability was significantly greater in older adults as shown in Chapter 4 (Figure 4-3). When all trunk state variables were considered here, the aggregate variability as defined here was not different, since the variability of other individual variables (other rotational angles and velocities in Chapter 4; Figure 4-3) were not age-dependent.

We used RMSD to adjust the variability measure by the size of the “mean,” rather than using coefficient of variation (CV), as a new way to normalize variability to the size of the dynamics. CV is defined as standard deviation divided by the mean, but can give spurious results if the mean approaches zero, or if the mean value of a variable crosses zero, which often occurs in biomechanics. Instead, we measured the RMS radius of the trajectory as a measure of the "mean" or the normalizing factor for variability. This avoids the problem of the zero-crossing. It also provides a measure of variability around an attractor (or the mean cycle) that considers the dynamics of all state-variables. Previous studies have noted that superior segments exhibit less variability of movement as disturbances are absorbed (Kavanagh, Morrison et al. 2006). Our results agree with these results, but if we account for the size of the motion the segments experience, the superior segments experience more normalized variability compared to inferior segments, whose variability relative to their size of motion is smaller (Figure 6-8).

The inferior segments exhibited smaller MeanSD/RMSD ratio, similar to having a larger signal-to-noise ratio (SNR) which may have affected the stability results. Would a more valid comparison be made between segments if all state spaces were normalized to RMSD? This does not seem to the case, since despite more relative variability, the superior segments were not overall more sensitive to perturbations. Also, the calculation of Floquet multipliers is not dependent on the size of the state space, only the relative size of each state variable. The results suggest that it is more important to study the dynamics of trunk motion during gait, as perturbations persist longer. Clinical interventions need to address the importance of the trunk segment during gait, such as developing tests of the functioning of trunk musculature, as well as interventions such as strength training.

Previous observations have noted that head movement is always tightly controlled during various tasks, such as standing, dynamic movements (Pozzo, Levik et al. 1995), and walking (Holt, Ratcliffe et al. 1999; Cromwell, Schurter et al. 2004). Shock absorption that occurs in inferior segments during walking results in only minimal influence on the motion of the head (Ratcliffe and Holt 1997). These observations suggest that one goal of the nervous system is to stabilize the head segment. It is hypothesized that lack of head control may indicate risk of falls (Holt, Ratcliffe et al. 1999). Control of head movement comes from compensating for trunk movements as well as movements of more inferior segments (Cromwell, Newton et al. 2002). We found that superior segments are more unstable, it is not clear how the stability of the head segment relates to the rest of the body.

Height-dependence of the stability of body segments was only investigated at the preferred walking speed. However, this relationship may change with different walking speeds, and should be investigated in the future. The capacity to deal with perturbations can be best observed at the edge of stability (Venkadesan, Guckenheimer et al, 2007). Observed segment and age effects may be more pronounced at fast speeds. Also, the efficacy of interventions that can help stabilize the trunk should be studied.

Chapter 7: The effects of aging and walking speed on the variability and stability of electromyography patterns

INTRODUCTION

To understand falls that occur during walking, it is important to study how the nervous system responds to perturbations during gait. Falls occur after a perturbation when the necessary corrective responses to counteract the effects of the perturbation are not provided. Normally, the neuromuscular system senses the perturbation, and activates the motor pathways, which in turn activate the muscles in order to produce corrective action. If any of these functions are impaired, through sensory loss, cognitive or motor impairments, or the loss of muscle function, a person may not be able to correct for the effect of the perturbation and a fall may occur. To facilitate stable walking, a person must generate both steady, correct motor patterns, and also the appropriate corrective responses to perturbations.

To understand how motor patterns are regulated during gait, we first need to understand how motor patterns, as described using surface electromyography (EMG), vary. Although kinematic variability is influenced by age and walking speed as shown in Chapter 4, not much is known about how variability of EMG activity is affected by these same factors. The only study that systematically explored this issue showed that slower walking is associated with both increased variability in amplitude and timing, and that aging increases the variability in amplitude of EMG (Shiavi, Bugle et al. 1987). The coefficient of variation (CV) of EMG signals in *vastus lateralis* and hamstrings muscles decreased with speed, but in *gastrocnemius* and *tibialis anterior* EMG signals did not change with speed. However, these CV's did not provide any information on the changes in EMG amplitude during different walking speeds. Slower walking is associated with increased variability of burst duration and onset latency (Chung and Guiliani 1997), but its effect on amplitude is not known. Also, because Shiavi *et al* used self-selected speeds such as "slow" and "fast" (Shiavi et al, 1987), without accounting for differences in body size or an individual's preferred walking speed. This made comparisons between age groups and speeds difficult. In this study, we addressed this by comparing the variability of EMG signals in both young and older adults across multiple controlled speeds using a treadmill.

Using variability measures to describe the characteristics of motor patterns during walking provides us the statistical properties of the fluctuations overall, but these methods ignore how individual fluctuations in motor outputs relate to each other from one stride to the next. In most studies, EMG signals are ensemble-averaged over multiple strides, ignoring the time-course of how EMG varies over many consecutive strides. These methods do not adequately describe the interaction of multiple muscles, or the fluctuations of motor patterns over time during walking. Often, signals from many muscles are studied only one at a time. The studies that use multivariate methods often do not adequately describe the coordination of muscles over time. Studies employing principal components analysis to describe the activity of multiple muscles at once (Ivanenko, Poppele et al. 2004; Ivanenko, Poppele et al. 2006) have not accounted for the stride-to-stride fluctuations in EMG. The same is the case for a multivariate clustering methods to describe walking uphill and on level ground (Jansen, Miller et al. 2003).

To address the limitations of previous studies, we can use a state-space description of EMG signals to describe walking, which will account for its multivariate and temporal behavior. Because of their multivariate, somewhat periodic nature, motor patterns during gait are well suited to be described using state-space methods. In particular, the robustness of motor patterns during walking can be assessed by quantifying the stability of motor outputs measured using electromyography to describe age-related changes in motor patterns. A state-space description allows tracking of cycle-to-cycle fluctuations of these activation patterns in multiple muscles over multiple strides. By tracking multiple muscles at once, state-space descriptions may be able to track changes due to aging, fatigue, or therapeutic intervention better than by studying the fluctuations only in individual muscles.

In this study, we quantified the stride-to-stride variability of EMG linear envelopes during gait as a function of walking speed and age, similar to Chapter 4, to see if age influences variability of EMG when controlled for speed. Also, local and orbital stability of EMG signals were also quantified to describe how the EMG amplitude patterns to change over multiple strides.

METHODS

Data Collection

Data were collected during the walking experiment as previously described in Chapter 3. Kinematics of the feet were used to track gait events. Muscle activation patterns were measured using surface electromyography (EMG). The bipolar electrodes were placed on 4 muscles of the left leg: *vastus lateralis* (VL), hamstrings group (HA), medial head of *gastrocnemius* (GA), and *tibialis anterior* (TA) according to the SENIAM conventions (Konrad 2005). The skin was prepared by shaving and using alcohol to remove excess dead skin and oils.

The recorded EMG signals were bandpass filtered (passband 20-300 Hz), notch-filtered at 60 Hz using a Butterworth filter, and demeaned. Then, signals were normalized to the peak amplitude during the preferred walking speed trial of each person, to allow for better comparison between subjects. Signals were then rectified, and low-pass filtered ("smoothed") by a moving average filter. The signal was convolved with a

Hamming averaging window of 100 ms, equivalent to a low-pass filter with 10Hz cutoff (Winter 1990). A Hamming window is a bell-shaped curve that calculates a weighted average, giving less weight to the ends of the averaging window. Once smoothed, the signal was down-sampled to 60Hz to determine the final linear envelopes. Any extraneous noise in the signal due to static discharge, electrode mal-adhesion, etc. was noted after a visual inspection. A noise vector was created to track when these extraneous noise events occurred, for each signal of each trial. In some subjects, the VL muscle was mostly quiescent during slow walking speeds. Such trials (15 out of 349 collected) were not included in the variability analysis for VL. The same selection was performed for the signals from HA (17/349), GS (8/349), and TA (7/349) muscles due to technical problems. Any trials with unusable EMG signal(s) (total 29/349) were also excluded in their entirety from the state-space analyses, as data from all 4 muscles were used for all trials.

Variability Analyses

Inter-stride variability of EMG signals was quantified using methods used in Chapter 4. The data for each stride during walking were normalized to 0-100% gait cycle. Means and standard deviations of linear envelopes were calculated at each percentage of gait cycle. To determine the variability of the linear envelopes over the entire gait cycle, the MeanSD was determined (Eqn. 7-1) (Dingwell and Marin 2006):

$$MeanSD = \langle SD(i) \rangle_i,\ i \in \{0-100\%\ \text{gait cycle}\} \qquad (7\text{-}1)$$

where $SD(i)$ indicates the standard deviation of a measure at ith % gait cycle, and $\langle\ \rangle_i$ denotes the average over all i. One MeanSD value was calculated for each muscle of each trial.

State Space Description of Segments

Previous work using principal components analysis have shown that a small number of signals can reconstruct most of the features of the EMG signals of multiple muscles during walking (Ivanenko, Popele et al. 2004). The use of principal component analysis to reduce multi-muscle EMG signals in human walking suggested that five principal components are enough to describe all of the muscle activation patterns at different walking speeds (Ivanenko, Poppele et al. 2004). This is supplemented by the global false nearest-neighbors studies, where 5 state variables are sufficient for describing the dynamics of the human walking (Dingwell and Cusumano 2000). These studies suggest that five state variables may be sufficient to describe and capture limit-cycle-like behavior of the muscle activation patterns during walking.

A state-space description of EMG signals was created using the four normalized (now unitless) signals as processed above, as well as their time derivatives, non-dimensionalized by $\sqrt{LL/g}$, where LL = leg length, to create an 8-dimensional unitless state-space (Packard, Crutchfield et al. 1980). The state-space constructed with time derivatives are in theory dynamically equivalent to other formulations of the state-space. Pilot work also showed that four principal components can be derived from this 8-dimensional state space (Appendix B). From the state-space description, the magnitude of the largest Floquet multiplers (FM) and mean divergence curves were calculated as described before.

Statistics

MeanSD from each muscle, Mean log divergence slopes λ^*_S, $\lambda^*_{L,}$ and FM at 0, 25, 50, and 75% of the gait cycle were compared between age groups and speeds using a repeated-measures ANOVA using SPSS 14 (SPSS, Chicago IL). Because of intra-subject variability of FM (Fig 7-3), intra-subject mean (MeanFM) values were also calculated and compared. The analysis was repeated as an ANCOVA, where the composite strength and ROM scores were included as covariates. Tukey's LSD tests were used for any post-hoc analyses.

RESULTS

Variability of EMG linear envelope amplitudes increased with walking speed in VL, HA and TA muscles ($p < 0.0001$). Variability was also larger in older adults in HA ($p < 0.004$) and TA ($p < 0.001$) muscles. Interaction effects were not significant. Variability of EMG was largely dependent on the amplitude of the signal. This exhibits a characteristic known as "signal-dependent noise" (Harris and Wolpert 1998), where the force variability from a muscle increases linearly with force magnitude. Mechanisms of signal-dependent noise in muscle force output have been explored, but not for EMG.

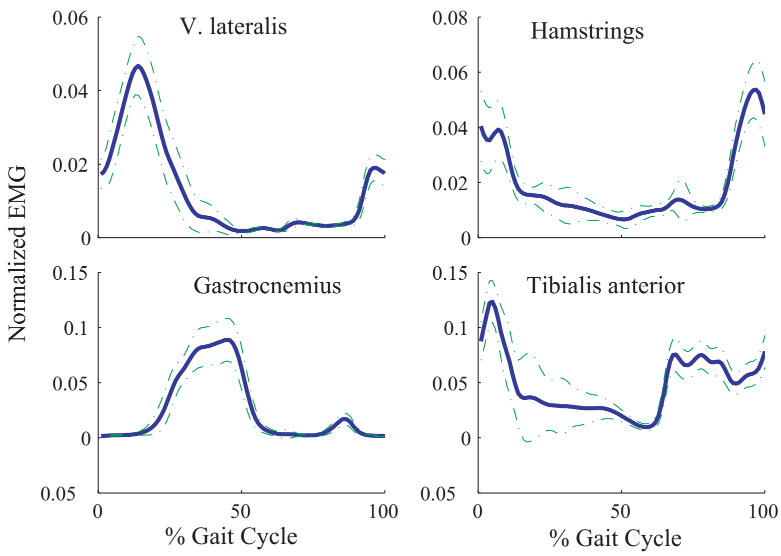


Figure 7-1. EMG Linear Envelopes

EMG linear envelopes from a single subject is shown. Blue solid line represents the inter-stride mean, and the green dashed line represents ± 1 S.D.

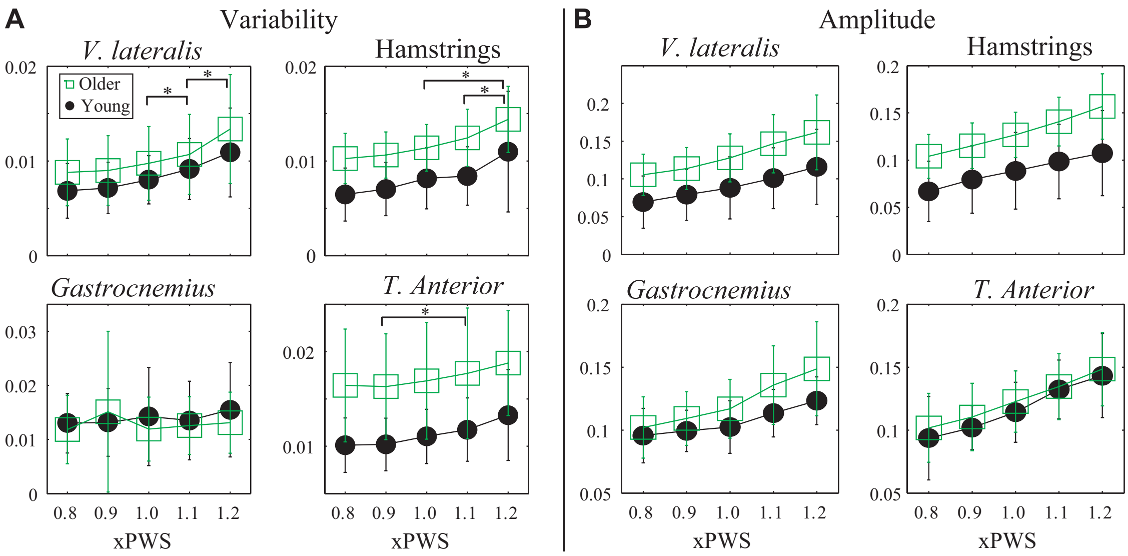


Figure 7-2. MeanSD and Peak Amplitude of EMG Envelopes vs. Speed

A: MeanSD of EMG envelopes. Speed effects were significant in all but the GA muscle. Age effects were significant in HA and TA. B: Between-strides average of EMG envelope peak amplitudes. Amplitudes increased with speed ($p < 0.001$). Older adults displayed higher amplitudes in VL ($p <0.003$), HA ($p < 0.001$), and GS ($p < 0.03$). When MeanSD was normalized by Peak EMG amplitudes, age- and speed- related differences no longer existed ($p > 0.05$). Horizontal brackets denote significant Tukey's LSD post-hoc comparisons at $p < 0.005$. Error bars denote standard deviations within each group.

Table 7. P-values for variability comparisons

	VL	HA	GS	TA
Age	0.182	0.0031	0.404	0.00098
Speed	6.78E-24	1.879E-18	0.0265	1.159E-10
Age x Speed	0.660	0.7418	0.5386	0.706

Sample FM data are shown in Figure 7-3. FM of older adults were larger at 0, 25, and 50% of the gait cycle ($p < 0.003$; Figure 7-3), while showing a similar trend at 75% ($p <0.009$). MeanFM was also larger in older adults ($p < 0.0001$). Speed effects were not significant, but interaction effects were significant at 25% ($p < 0.0001$), where speed effects were seen only in young adults ($p < 0.003$), and in MeanFM ($p < 0.0001$).

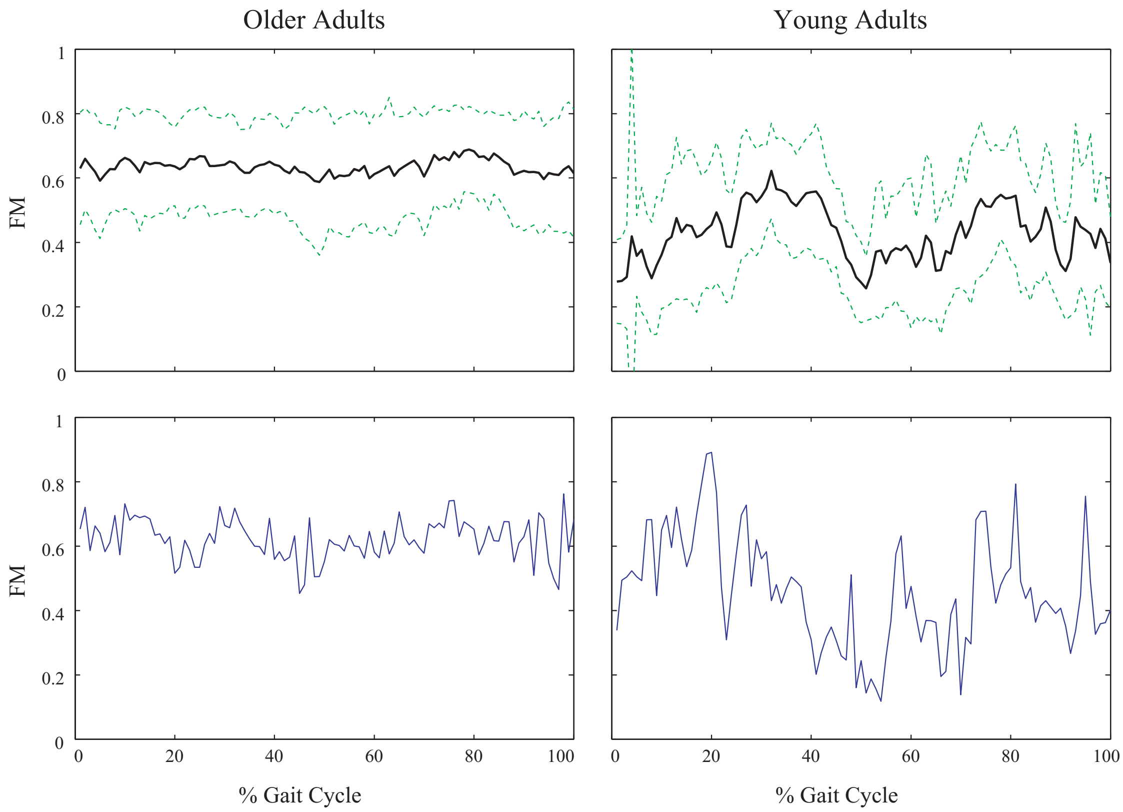


Figure 7-3. Floquet Multipliers across the cycle.

FM values are displayed for the 1x PWS speed. Top: Group averages with standard deviation bars (dotted line). Bottom: A typical trial from each group (left: older adult, right: young adult). FM varied much more across the gait cycle in young adults, but it was not very consistent across subjects, while FM was consistent across the cycle in older adults. A few older adults displayed a pattern similar to young adults, but there was no obvious reason why. Although this may be an indication of a young-adult like motor behavior, there were no immediately obvious reason for this phenomenon.

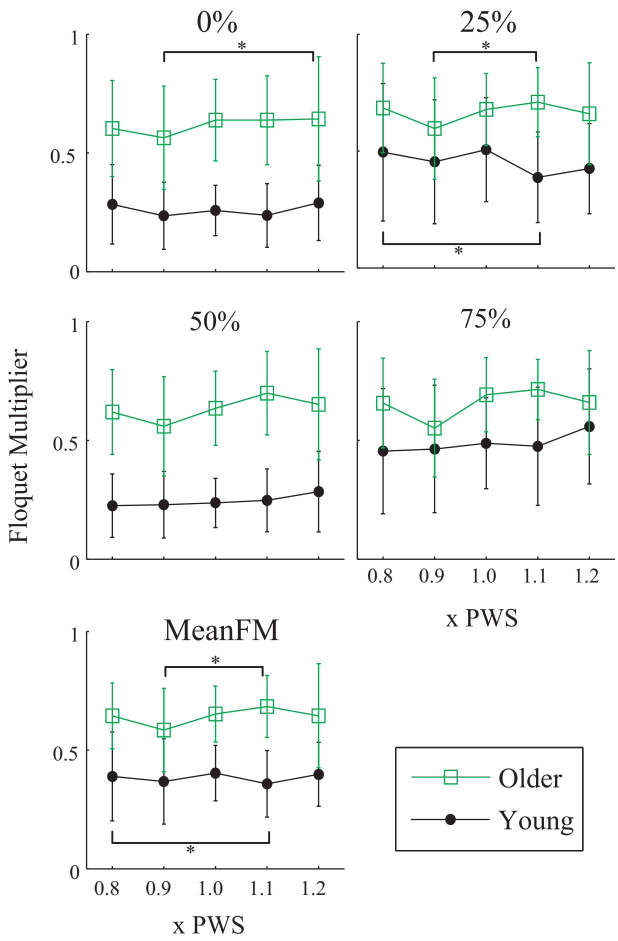


Figure 7-4. FM vs. Speed.

FM for 0, 25, 50, and 75% of the gait cycle are listed. Because of intra-subject variability of FM, intra-subject mean (MeanFM) values are also presented. Main effect for Age was significant for all except at 75% (see text). Error bars denote standard deviations within each group. Horizontal brackets denote significant Tukey's LSD post-hoc comparisons at $p < 0.005$. When they appear both at top and bottom, they denote the post-hoc comparisons for each age group separately.

Mean log divergence curves showed a sharper rise in the beginning compared to those calculated from kinematics, as shown in Chapter 5 (Figure 7-5). Both divergence exponents increased with speed ($p < 0.00001$), while λ^*_S was greater in older adults ($p < 0.003$), similar to the findings in Chapter 5 (Figure 7-6). The EMG amplitude pattern was more sensitive to perturbations in older adults. When adjusted for strength or ROM, the age-related differences were no longer significant (Table 8). Their relationship is shown in Figure 7-7.

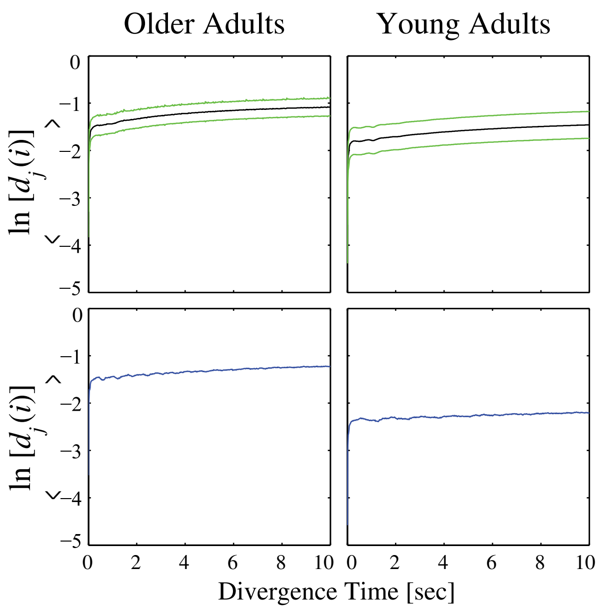


Figure 7-5. Sample divergence Curves.

Top: Group means and standard deviations at 1x PWS speed.
Bottom: Mean log divergence curve from a representative trial.

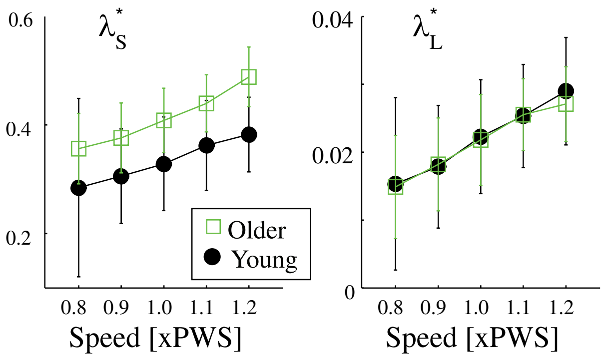


Figure 7-6. Relationship of divergence exponents and gait speed

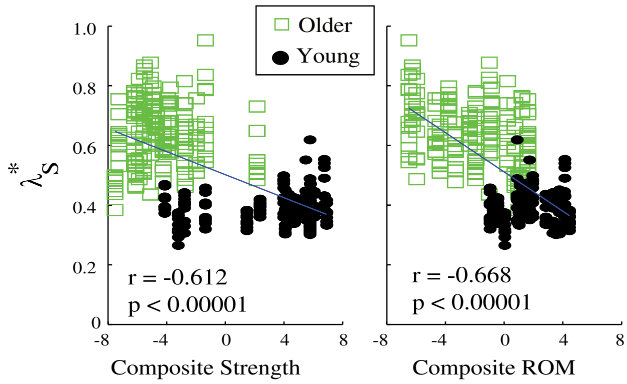


Figure 7-7. Relationship of Strength and ROM vs. short-term divergence exponent

Table 8. P-values for stability comparisons

p-values	λ^*_S	λ^*_L	0%	25%	50%	75%	MeanFM
Age	0.0020	.979	<0.00001	0.00236	<0.00001	0.00830	<0.00001
Segs	<0.00001	<0.00001	0.1650	0.190	0.1838	0.230	0.252
Age x Speed	0.757	.990	0.0246	<0.00001	0.577	0.0263	<0.00003
Age, adj for Str.	.0517	N/A	<0.00005	0.02693	<0.00001	0.1173	<0.00004
Age, adj. for ROM	0.192	N/A	<0.00001	0.05278	<0.00001	0.0321	<0.00002
Age, adj for both	0.382	N/A	0.000194	0.10600	<0.00001	0.147	0.00013

DISCUSSION

To understand falls in the elderly, we need to understand how aging affects motor patterns during gait. Fluctuations in kinematics may reflect the health of the motor system (Hausdorff, Mitchell et al. 1997; Hausdorff, Ashkenazy et al. 2001), but the little is known about how these fluctuations occur and are controlled. Since the nervous system generates the motor patterns that result in the observed kinematics, we need to study how aging affects the variability of motor patterns. Because gait exhibits a near-cyclic behavior, and fluctuates over time, we need to account for the time-course in which these fluctuations occur. These fluctuations traditionally have been quantified only as variability, studying the spread about the mean, but these measures fail to account for the time-course. By using state-space methods that account for this, we can better understand the effect of aging on motor patterns during gait, and thereby understand how they manifest in kinematic fluctuations.

Variability of EMG patterns during gait increased with speed, except in the GA. Age-differences were seen only in HA and TA. This behavior is unlike the response to walking speed in kinematic variability of the leg joint angles as shown in Chapter 4. Even though these muscles are involved in sagittal motion, the variability of sagittal joint angles did not exhibit this speed-dependency or age-effect. This suggests that variability of EMG patterns do not predict that of kinematic variability in response to walking speed.

What caused these age-related changes in variability? Variability in EMG can come from many sources. Burst duration and onset latency became more variable with slower walking in older adults (Chung and Giuliani 1997). The loss of motor units with age results in larger motor units due to increased innervation of low-threshold motor units (Kanda and Hashizume 1989; Kadhiresan, Hassett et al. 1996). The larger motor units would produce larger EMG signals, would amplify the effect of increased variability in timing. In healthy young adult men, no relationship was found between the amount of motor unit synchronization in various leg muscles and the speed of walking (Hansen, Hansen et al. 2001). However, this finding may not extend to older adults, where any changes in the amount of synchronization may influence the observed variability of EMG amplitudes. In future work, motor unit synchronization in large muscles in older adults may help explain these age-related differences. Also, the effect of strengthening exercise interventions on motor unit synchronization as well as EMG activity need to be investigated.

EMG in older adults exhibited less orbital stability, similar to results found in Chapter 5. This indicated that any disturbed motor patterns return toward an average pattern slower, where effects of the disturbed motor pattern linger over more strides, compared to young adults. This could be because it takes them longer to recover after a perturbation, and may explain the kinematics results shown in Chapter 5. Speed effects were not very strong, but showed some tendency toward being more unstable at faster speeds.

Similar to Chapter 5, λ^*_S was found to be larger in older adults. This indicates that compared to one motor pattern, slightly different motor patterns would diverge away (or become different) at a faster rate in older adults, and were more sensitive to perturbations. In terms of dynamics, this could be described as the attractor becoming weaker, and not being able to pull nearby orbits back toward itself as well. However, biological reasoning for this phenomenon is more difficult to establish. Repetitive coordinated muscle activity in vertebrates can be attributed to spinal central pattern generators (CPG) (Shik and Orlovsky 1976). This weakened attractor may be reflective of the deterioration of CPG. The inability to return toward the attractor also reflects inability to correct perturbations promptly. Reflex mechanisms to correct for stumbling described by Dietz, Berger and Quintern (1986), may be deteriorated in older adults, these mechanisms have not been studied in older adults. Future work should explore the relationship between this mechanism and stability.

We have described the age- and speed-related differences in EMG variability during gait, as well as the differences in response to disturbances in motor patterns using state-space methods. In future work, we need to study how interventions for reducing fall-risk in the elderly, such as strength training, affects nervous function during gait. We did not control for the types of perturbations that may have caused various disturbances in the motor patterns, which may have come from external mechanical sources, motor errors, or neural noise. Future work will also have to distinguish the effects of different perturbation types such as mechanical perturbations, dual task, and nerve blocks.

Chapter 8: Conclusions and Future Work

SUMMARY AND CONCLUSIONS

In order to understand falls in older adults that occur during walking, it is important to understand the strategies used by the nervous system to maintain stability, and how aging affects walking. The major age-related changes can be categorized into the following groups: physical changes such as muscle weakness, reduced range of motion, and the decline of sensory function; and behavioral changes such as slower walking speed, increased double-support phase, increased variability (Winter, Patla et al. 1990; Bassey, Fiatarone et al. 1992; Dobbs, Lubel et al. 1992; Maki 1997). The causes of these behavioral changes have not been established. The difficulty lies in separating possible underlying causes.

Among the behavior changes with aging, gait variability has received much attention because increased variability has been shown to be related to aging and fall risk (Lord, Lloyd et al. 1996; Maki 1997; Hausdorff, Rios et al. 2001). Older adults display higher gait variability compared to healthy young adults (Owings and Grabiner 2004), but the cause of this higher variability is unclear. Older adults typically walk slower (Alexander 1996), but healthy young adults also become more variable as they walk at slower speeds (Winter 1983; Dingwell and Marin 2006). This suggests that increased variability observed in older adults may be a result of slower walking speed. Clarifying this issue involves separating the effects of walking speed on gait variability from that of age. In Aim 1 of this project, we established that greater gait variability exists in healthy older adults separate from slower walking speeds, and this age-related difference can be explained by diminished strength and range on motion in the lower extremity. This is the first study that separated the issue of aging from speed, and provided an explanation for the change associated with aging. The results suggest that strengthening and stretching interventions may help with gait variability and fall risk. However, a strengthening intervention had only a small effect on gait variability, even though it reduces fall risk (Hausdorff, Nelson et al. 2001). It is not clear why and needs to be investigated further.

Why do older adults walk slower? Some have suggested that age-related physical factors such as muscle weakness and stiffness may force people to walk slower, and put them at risk for falls (Larish, Martin et al. 1988; Elble, Thomas et al. 1991). Others suggested that people *choose* to slow down to allow for more double-support and stability (Murray, Kory et al. 1969; Winter, Patla et al. 1990). This disagreement in the literature can be resolved by quantifying how stability varies with walking speed in older adults. In Aim 2, we demonstrated that the healthy older adults in this study exhibited more stability at slower walking speeds. They also preferred to walk at speeds comparable to young adults despite their decreased strength and range of motion. This leads us to conclude that weakness and stiffness did not force these healthy older adults to walk slower, and therefore it is more likely that they choose to walk slower to improve stability. We have provided the first evidence to answer why healthy older adults walk slower. However, more work is required to understand the issues of slower walking in clinical populations.

Some have suggested that the goal of postural and locomotor stability systems may be to maintain superior segment stability, and inferior segments work to maintain the stability of superior segments (Pozzo, Levik et al. 1995; Holt, Ratcliffe et al. 1999;

Cromwell, Schurter et al. 2004). In Aim 3, we explored the relationship of body segment height and stability during gait. We discovered that superior segments exhibit less orbital stability during preferred walking speed, in contrast to previous suggestions otherwise. This suggests that the trunk segment is more susceptible to perturbations, and clinical interventions need to address trunk stability. Development of appropriate interventions is indicated.

Motor function is also affected by aging. Fluctuations in motor activity during walking are not well understood, especially in the context of aging. Higher force variability found in older adults may indicate increased motor errors (Tracy and Enoka 2002), but age-related characteristics in motor behavior have not been quantified in depth during gait. Previous methods generally consisted of studying single muscles one at a time, not accounting for the time-course in which fluctuations occur. In Aim 4, we used state-space methods to quantify the stride-to-stride fluctuations of muscle activation patterns during gait. Variability increased with speed except in the *gastrocnemius* EMG, and only hamstrings and *tibilalis anterior* muscle EMG signals were more variable in older adults. Orbital stability of EMG signals was less in older adults, suggesting that a disturbance in a motor pattern takes longer to return toward the average pattern. Neighboring trajectories diverge away faster in older adults, suggesting that their motor patterns are more sensitive to perturbations. These results suggest that older adults have a diminished ability to tolerate disturbances of motor patterns during gait.

Looking at these together, we draw the following conclusions. First, although the trunk segment is the most massive, it is less stable than inferior segments. These results suggest that its control may be more important for preventing falls during walking.

Second, deficits in strength and flexibility explain the age-related differences in gait variability and stability, when adjusted for speed. It is not yet clear if strength and flexibility training would reduce gait variability or improve gait stability. Third, the speed-dependency of stability is present in kinematics, but not in EMG. This suggests that the speed dependency in kinematic stability may come from mechanical sources, rather than neural sources.

DISCUSSION AND FUTURE WORK

Finding predictors for falls

It is often unclear what variables should be considered when studying variability or stability. In the literature, various measures are tested against fall risk, until a good predictor of fall risk is found. Step width variability and the Berg Balance Test are examples. In Chapter 4, variability of many different kinematic measures was tested, because there was no *a priori* basis for using a particular variability measure. Bonferroni corrections, which are very conservative, were used to account for multiple comparisons. Nevertheless, age- and speed-related differences were found. Aging may lead to an increase in variability, but increases or decreases in variability may depend on which variables are being examined and the context (Marin 2004). Since we did not study fall risk in association with these measures, it is not clear which of them may be related to fall risk.

We identified gait variables whose variability is more sensitive to the effect of age, even when controlled for speed, from others whose variability is sensitive mainly to the walking speed. Trunk roll and pitch, and stride time variability may be indicative of aging process that related to fall risk. These variables may be more sensitive to the diminishing motor control associated with aging, and thus may help us better identify fall risk, and better monitor improvements in motor control as a measure of efficacy of interventions to prevent falls.

Effects of Age and the Sources of Variability

We have not investigated fully the mechanistic reasons behind increased variability in the elderly. We have demonstrated that speed by itself is not, but decreased strength and ROM are correlated to decreased variability. We have yet to determine what it is about loss of strength or flexibility that leads to variability.

Variability of gait comes from many different sources, such as internal sources (natural variation, aging effects, pathological mechanisms), and external sources (mechanical perturbations, instrumentation and methodology, and the environment) (Chau, Young et al. 2005). In this study, we controlled for most of these factors, in order to describe age-related effects. However, we did not directly explore the mechanistic basis for these age-related changes. The increased variability may come from loss of motor units, and associated increase in force unsteadiness during isometric tasks. Older adults display more force unsteadiness, which may be caused by increases in force produced by a single motor unit, due to increased innervation of low-threshold motor units (Kanda and Hashizume 1989; Kadhiresan, Hassett et al. 1996). However, but larger MU forces in FDI muscle in older adults were not associated with larger synchronization (Semmler, Steege et al. 2000), which does not explain the increased variability. Strength training seems to reduce the variability of firing rates (Duchateau, Semmler et al. 2006), which may possibly explain the slight reduction of gait variability after strength training (Hausdorff, Nelson et al. 2001), but more work is required. Motor unit synchronization findings in isometric studies may extend to dynamic tasks during walking (Hansen, Hansen et al. 2001), but this synchronization has not been investigated in depth. Before we can relate single-motor unit level findings to variability in gait, we need to investigate whether the loss of motor units and motor unit synchronization during gait in the larger muscles involved. More work in this area will help to clarify the sources of gait variability due to the muscles and other effectors of the nervous system, separating them from other sources, such as sensory function, frontal executive function of the brain, and sensory integration.

Measuring Stability

We have demonstrated the age and speed effects on two dynamic stability measures of gait. These measures describe two different aspects of the dynamics of the system. These local and orbital stability measures are designed to study deterministic systems whose behavior is explicitly defined mathematically. They were not designed to describe behaviors that show natural variability (such as gait). Thus they cannot be interpreted in the same manner as if human gait follows some explicit mathematical formula. Otherwise, erroneous interpretations can be made, such as claiming that human walking exhibits deterministic chaos (Miller, Stergiou et al. 2006), even though such terms cannot be applied to human walking. Nevertheless, these measures describe the dynamics of human walking, and provide insights into how the locomotor system deals with perturbations that measures of variability cannot. We have described how the body responds to perturbations and corrects for them, and thus provided a deeper view into the dynamics of gait.

Using the current methods, we did not find any consistent variations in orbital stability across the gait cycle. This result was somewhat expected, as theoretically Floquet multipliers does not vary across the cycle. However, systems do exist that display differing amount of local stability and instability (Ali and Menzinger 1999), and the current methods were not able to quantify this behavior during walking. Nevertheless, the evidence suggests that the double-support phase of the gait cycle is more stable. Therefore, other methods are required to answer this question. Methods of Ali and Menzinger (1999) have yet to be adapted in the context of walking. Also, human walking exhibits both deterministic and seemingly stochastic processes. Methods that can describe stability in systems with noise or inherent variability would further the understanding of dynamic stability during gait.

Appendices

APPENDIX A. HEALTH-HISTORY QUESTIONNAIRE

HEALTH HISTORY QUESTIONNAIRE
"Local Dynamic Stability of Walking and Young and Elderly Adults"

IRB #: 2005-03-0013 Subject ID: ___________
Date of Birth (mm/dd/yy): ________________________________ Age: ________________ ________

MALE: ________ FEMALE: ________

Height: ________________ ft./in. = __________ in. × 0.0254 = _____________ m

Weight: ________________ lbs. × 0.4567 = ____________ kg.

BMI (kg/m^2): ________________ (BMI > 35 excludes)

1. Are you taking any medications on a regular basis? Y / N
(Exclusions include: Psychotropics, Antihistamines, Asthma Meds, Aldomet, Clonidine, Anti-Depressants, Anti-Anxiety Meds)

2. Any over- the -counter meds? Y / N
If yes, explain:

3. Do you have any disability or impairment that affects you when you walk? Y / N
(If yes, excludes.)

4. Have you had any broken bones, surgery, or injury to lower extremities? Y / N
If yes, explain:

5. Do you have arthritis? Does it cause pain or discomfort when you stand or walk? Y / N
If yes to discomfort, excludes.

6. Have you had any significant medical problems within the last 10 years? Y / N
If yes, explain:

7. Do you have a history of neurological diseases likely to affect your ability to stand or walk, including CVA (stroke), disc disease, peripheral neuropathy, or lower extremity weakness? Y / N
If yes, exclude.

8. Do you have any history of back problems, such as low back pain? Y / N
If yes, explain.

9. Do you have any problems with standing balance? Y / N
If yes, excludes.

10. Do you have any drug and/or alcohol dependence? Y / N
If yes, excludes.

11. Do you have any significant visual impairments? Y / N
Examples: loss of binocular vision or the presence of double vision
If yes, excludes.

12. Do you have any heart problems or coronary artery disease? Y / N
If yes, excludes.

13. Do you have hypertension? Y / N
If yes, excludes.

15. Do you have any lung or respiratory problems? Y / N
If yes, excludes.

16. Do you smoke? Y / N
Pattern?

17. Do you use alcohol? Y / N
Pattern?

18. Do you use caffeine (cola, coffee, etc.)? Y / N
Pattern?

19. Do you have any allergies that require medication? Y / N
If yes, explain.

Self-reported activity level:

How many times a week do you exercise?: ______________________________

How long do you spend exercising on those days?: ______________________________

What intensity level would you say you exercise at?: ______________________________
(e.g. "low", "moderate", or "hard")

APPENDIX B. PRINCIPAL COMPONENT ANALYSIS ON EMG LINEAR ENVELOPES

Using EMG data, a pilot study was conducted to see if EMG signals have low-dimensional structure that would be suitable for a state-space analysis. If EMG does not have a low-dimensional structure, i.e., cannot be explained using a reasonable number of state variables, then a state-space analysis would not be appropriate.

The literature suggests that EMG patterns during walking can be divided into 5 principal components, or 5 separate synergies. (Ivanenko, Poppele et al. 2004; Ting and Macpherson 2005; Ivanenko, Poppele et al. 2006), suggesting there are 5 separate activation patterns that combine to form the activation pattern of each muscle.

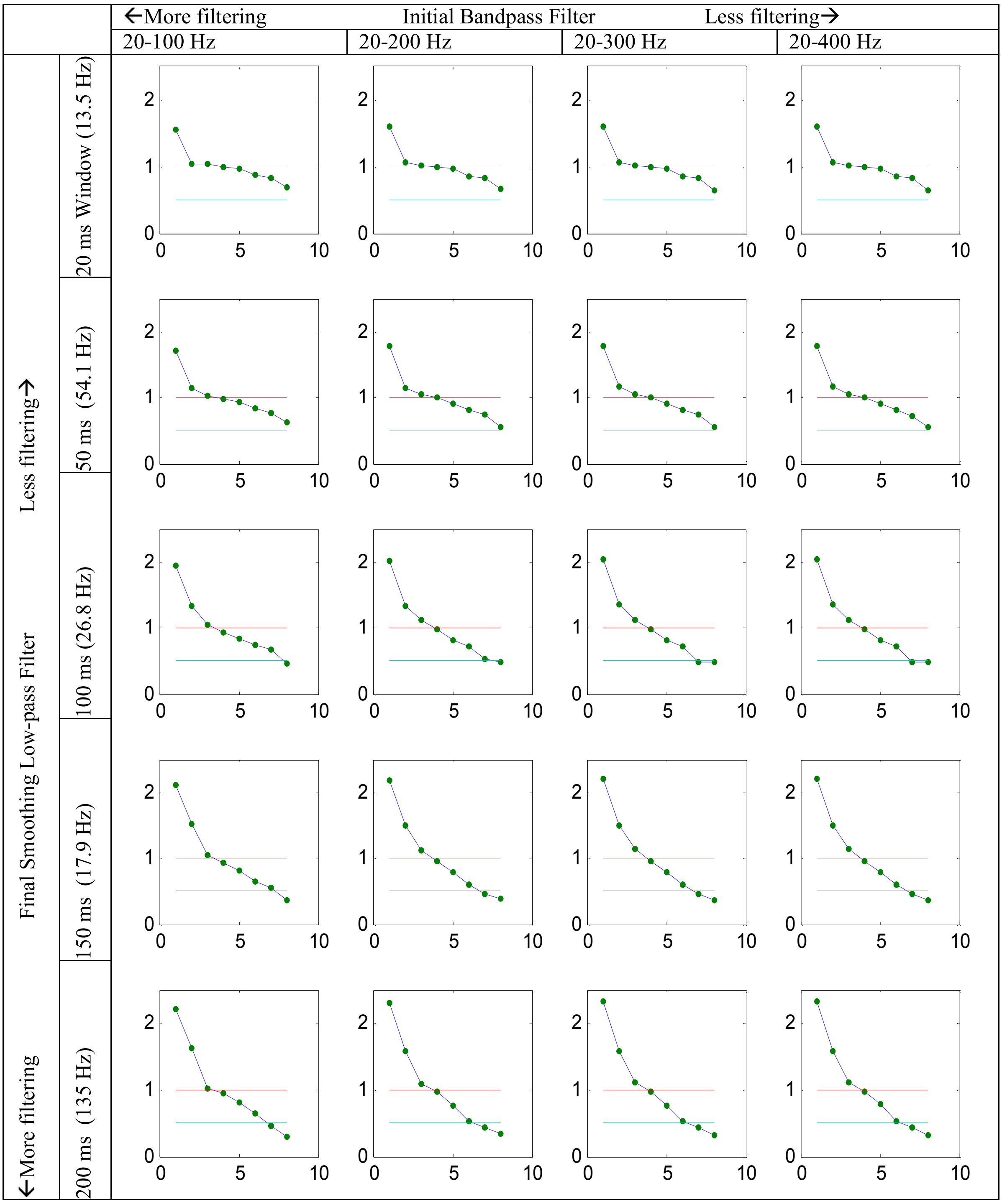
The four EMG signals from one walking trial from one subject were used in this pilot study. After filtering, rectification, smoothing and downsampling to 120 Hz, the 1st time derivatives of the signals were calculated. Smoothing was performed using a Hamming convolution filter that takes a weighted average of nearby values. Each of the 8 signals (4 processed original EMG signals and 4 of the derivatives) was demeaned and normalized to its own unit variance.

Principal Component analysis was performed via eigenvalue decomposition of the covariance matrix of the 5 minutes of walking data.

Experimental Design:
The robustness of the number of principal components derived from the data was studied as a function of filter cut-off frequencies and amount of smoothing.

The passbands studied were: 20-100Hz, 20-200Hz, -300 Hz, - 400Hz
The smoothing window size (Hamming) were: 20, 50, 100, 150, 200 ms, which have effective low-pass cut-offs of: 135 Hz, 54.1 Hz , 26.8 Hz ,17.9 Hz ,13.5 Hz

Number of Principal Components:
The Scree test simply looks for a kink on the Scree plot, where the subsequent principal components no longer explain the variance in the data. This is done visually.
The eigenvalue test keeps all the components with eigenvalue >1. A component with Eigenvalue <1 explains less of the variance in the data than a single original variable. A cutoff of 0.5 has been used in the literature



Scree Plots of Eigenvalues at various filtering parameters Horizontal line indicates eigenvalue = 1 and 0.5

Upon visual inspection, the eigenvalues do not follow the strict Scree test criteria. However, the eigenvalue plots do not change substantially across different filtering parameters

Using the eigenvalue test (at cutoff 1), we get the following number of principal components:

	20-100Hz	-200Hz	-300Hz	-400Hz
20 ms	3	4	4	5
50 ms	3	4	4	4
100 ms	3	3	3	3
150 ms	3	3	3	3
200 ms	3	3	3	3

Using the eigenvalue test (at cutoff 0.5), we get the following number of principal components:

	20-100Hz	-200Hz	-300Hz	-400Hz
20 ms	8	8	8	8
50 ms	8	8	8	8
100 ms	7	7	6	6
150 ms	7	6	6	6
200 ms	6	6	6	6

Reasonable filter cut offs are indicated by bold text.

Conclusion:
The number of principal components identified with the eigenvalue test (= 1) seems robust to changes in EMG processing parameters. Although the higher amount of smoothing 100-200 ms) seems to lower the number from 4 to 3, the eigenvalue of the 4^{th} component is still very close to 1 in both groups of cases.

APPENDIX C. STRENGTH AND RANGE OF MOTION RESULTS

	Hip Flexion		Hip Extension		Knee flexion		Knee Extension		Ankle Plantar-Flexion		Ankle Dorsi-Flexion	
Subject	R	L	R	L	R	L	R	L	R	L	R	L
Y5	149	137	0	0	142	141	0	0	65	62	30	32
y6	141	143	0	0	142	150	0	0	69	72	10	7
y7	150	132	0	0	140	139	0	0	72	64	20	26
y8	149	148	0	0	142	132	0	0	66	75	20	16
y9	153	138	0	0	144	143	0	0	73	60	19	23
y10	152	136	0	0	148	146	0	0	84	76	15	11
y11	150	136	0	0	146	147	0	0	62	63	17	15
y12	147	137	0	0	143	149	0	0	59	50	11	19
y13	140	138	0	0	139	142	0	0	59	61	13	11
y14	148	148	0	0	149	155	0	0	65	59	14	14
y15	139	144	0	0	145	140	0	0	65	62	24	10
y16	140	137	0	0	145	149	0	0	55	40	10	12
y17	140	141	0	0	141	149	0	0	51	45	23	25
y18	141	135	0	0	146	152	0	0	44	51	4	3
y19	137	131	0	0	142	136	0	0	67	75	15	9
y20	141	142	0	0	140	139	0	0	58	66	2	3
y21	139	134	0	0	134	138	0	0	80	82	18	8
mean	144.5	138.6	0	0	142.82	143.94	0	0	64.35	62.53	15.59	14.35
SD	5.375	4.999	0	0	3.6269	6.2896	0	0	9.937	11.46	7.089	8.246
E7	139	136	0	0	136	149	0	0	57	48	20	15
E8	131	135	0	0	145	143	0	0	47	42	18	17
E9	118	118	0	0	128	132	-5	-5	59	53	16	13
E10	130	128	0	0	146	150	0	0	67	59	12	15
E11	137	125	0	0	148	148	0	0	52	59	3	5
E12	139	139	0	0	145	148	0	0	62	57	20	21
E13	138	136	0	0	139	137	0	0	71	51	8	8
E14	140	133	0	0	144	144	0	0	62	69	29	25
E15	136	137	0	0	137	136	0	0	62	65	21	15
E16	118	113	0	0	136	136	0	0	53	53	23	18
E17	119	114	0	0	155	133	-5	-5	39	41	17	7
E18	128	131	0	0	138	148	0	0	57	52	18	15
E19	119	117	0	0	135	128	0	0	46	46	10	5
E20	139	134	0	0	138	145	0	0	57	57	9	9
E21	132	133	0	0	148	147	0	0	62	66	20	19
E22	130	126	0	0	139	142	0	0	58	45	14	3
E23	132	142	0	0	139	146	0	0	61	52	14	13
E24	117	115	0	0	128	134	0	0	77	67	3	5
mean	130.1	128.4	0	0	140.22	141.44	-0.556	-0.556	58.28	54.56	15.28	12.67
SD	8.415	9.363	0	0	6.9414	6.8961	1.617	1.6169	8.989	8.521	6.867	6.269
P-value	1E-06	4E-04			0.1732	0.2708	0.163	0.1631	0.067	0.027	0.896	0.503

Right Leg, Forces (kg-f)

Subject	Knee Extension		Hip Extension		Knee flexion		Hip Flexion		Ankle Dorsi-Flexion		Ankle Planar-Flexion	
Trials	1	2	1	2	1	2	1	2	1	2	1	2
Y5	32.1	39.1	47.3	44.7	40.9	33	36.9	31.8	25.4	22.5	56	56.1
y6	42.1	41.9	37.5	36.1	23.4	22	36	38.8	17.6	17	42.8	48.7
y7	35	39.1	37.8	39.5	30.8	30.4	42.5	39.8	23.1	25.4	57.4	55.8
y8	33.6	37.4	39.2	40.5	37.8	39.2	36.4	36.4	23.9	24.2	56.5	63.8
y9	24.8	26.8	35	37.8	25.6	25.4	26.8	26.8	15.6	19.4	47.9	51.5
y10	23.4	23.2	23.7	23.5	20.3	16.6	21.1	20.1	15.6	16.6	48.2	52
y11	37.8	35.8	32.2	33.2	28.4	25.6	40.9	37.4	22.9	21.7	48.7	44.2
y12	33.5	36.1	42.2	44	36.4	38.3	37.8	35.2	27.1	27.1	54.9	47.3
y13	28.8	30.8	32.7	28.2	22.5	27.5	30.1	27	25.6	26.8	47.6	50.4
y14	24.9	26.2	37.4	38.6	30.1	33.3	33.6	29.4	19.4	19.7	51.6	52.3
y15	23.9	23.9	29	29.8	21.8	27.7	29.9	27.6	18	20.8	39.7	38
y16	39.8	32.7	37.1	32.6	25.2	20.9	36.4	32.4	25.3	22.8	49.8	50.4
y17	27.7	27.3	36	33.6	27.4	24.2	33.5	33.9	22.6	18.7	46.5	42.2
y18	37.5	33.8	30.2	31.9	23.1	26	40.3	39.2	22.6	26.5	44	43.4
y19	31.9	34.6	37.4	35.8	33.9	33.5	28.5	27.1	18.1	20.1	55.8	46.7
y20	29.9	31.3	30.2	27.6	22.3	25.7	23.1	23.1	21.2	19.4	42.2	40.8
y21	26.5	28.4	18.7	22.6	15.6	15.2	22.5	24.5	14.2	14.5	38	39.1
mean	31.36	32.26	34.3	34.1	27.38	27.32	32.72	31.2	21.1	21.36	48.7	48.39
SD	5.804	5.642	6.77	6.47	6.833	6.753	6.625	6.07	3.98	3.75	6.07	6.776
E7	20.9	23.7	19.8	19.4	17.2	16.9	23.7	25.9	16.3	13.9	39.7	35.5
E8	25.1	22.6	23.4	27.3	17.3	18.3	26	25.6	23.9	18.4	33	35.7
E9	30.4	29.9	33	32.9	20.4	20.8	24.6	22.6	16.3	19.3	40.9	44.5
E10	19.2	19.5	20.3	17.5	11.9	13.9	15.9	17.2	10.2	12.2	28.5	32.7
E11	30.7	23.4	27.7	29.8	19.4	21.5	28	30.5	33.5	36.1	16.1	18.4
E12	18.9	19.5	18.6	20	12.4	13.3	18	17.8	13.5	13.3	33.6	33
E13	24.9	24.5	19.4	21.5	11	12.5	18	20.8	8.5	8	27.1	27.4
E14	19.5	22.1	15.3	17	11.9	11	18.9	18.4	11.9	12.1	27.7	32.2
E15	24.5	21.2	19.7	19.5	17.5	17.6	18.9	20.4	15.5	14.4	31.9	29.1
E16	38.8	35.8	33.8	27.6	23.9	28.7	24.6	25.9	21.4	20	41.4	42.2
E17	25.3	23.7	22	22	16.3	15.6	20.6	23.4	16.4	17.3	32.4	34.4
E18	27.1	28.4	25.7	28.2	22	23.1	24.6	24	18	19.4	33.8	40.6
E19	32.1	30.4	22.5	23.5	22.6	20.4	16.1	19	20	19.7	34.9	38.4
E20	29.4	35.7	36.9	36.4	24.3	30.4	24.5	26.3	21.8	17.5	43	38.3
E21	27.4	26.5	26.5	26.2	20	20.1	26.3	24.3	17.2	14.4	36.7	37.5
E22	26.2	22.9	21.5	22	13.3	14.7	21.4	22.9	10	12.1	39.8	33.9
E23	29.3	27.3	25.7	27.4	21.7	22.3	29.4	30.4	17.5	17	41.6	40.2
E24	41.7	44.3	31.8	32.1	23.7	28.4	23.7	26.2	24.6	20.4	49.5	40.8
mean	27.3	26.74	24.6	25	18.16	19.42	22.4	23.4	17.6	16.97	35.1	35.27
SD	6.202	6.512	6	5.61	4.539	5.695	4.06	3.95	6.1	5.915	7.59	6.179
P-value	0.053	0.011	0	0	7E-05	8E-04	8E-06	0	0.05	0.013	0	1E-06

Distance to the force application from the joint center (cm)

Subject	Knee Extension	Hip Extension	Knee flexion	Hip Flexion	Ankle Dorsi-Flexion	Ankle Planar-Flexion
Y5	38	76	39	40	16	15
y6	38	81	39	40	15	14
y7	37	77	38	34	15	14
y8	41	78	40	40	16	14
y9	44	85	43	43	15	14
y10	38	74	37	41	13	13
y11	40	83	39	41	16	15
y12	45	89	45	46	15	16
y13	36	76	36	38	14	14
y14	37	75	37	41	14	14
y15	38	76	39	39	14	14
y16	43	89	43	46	15	15
y17	38	81	39	42	14	13
y18	43	89	44	44	14	15
y19	40	78	39	43	13	12
y20	40	79	40	41	14	14
y21	35	71	36	37	12	11
mean	39.47059	79.82353	39.58824	40.94118	14.41176	13.94118
SD	2.896499	5.51402	2.693947	3.051036	1.121318	1.197424
E7	37	78	38	40	13	12
E8	38	84	39	43	15	14
E9	36	76	37	37	13	13
E10	32	67	33	34	13	13
E11	44	87	44	45	14	15
E12	37	74	36	36	13	12
E13	39	81	39	37	15	13
E14	36	73	35	35	14	13
E15	39	79	39	39	12	12
E16	36	79	36	39	14	14
E17	40	85	42	43	15	15
E18	37	78	38	37	14	14
E19	39	86	41	44	15	15
E20	41	87	42	45	15	15
E21	40	79	40	39	15	14
E22	41	82	42	46	15	14
E23	37	76	38	37	14	14
E24	38	85	39	46	14	14
mean	38.16667	79.77778	38.77778	40.11111	14.05556	13.66667
SD	2.617812	5.429284	2.798225	3.998366	0.937595	1.028992
P-value	0.1727	0.98043	0.388989	0.493522	0.317193	0.47348

Left Leg Forces (kg-f)

Subject	Knee Extension		Hip Extension		Knee flexion		Hip Flexion		Ankle Dorsi-F		Ankle Planar-F	
Trials	1	2	1	2	1	2	1	2	1	2	1	2
Y5	36	33.3	40	44.7	41.7	36.7	31.5	31.9	24.6	25.6	54	61
y6	38.6	34.6	31.8	30.8	27.3	22	38.4	36.7	16.3	16.3	49.9	46.8
y7	34.1	32.9	40.6	41.4	26.3	29.8	36	39.4	27.6	29.8	58	55.1
y8	29.1	34.6	39.2	30.3	36	34.7	36.7	37.1	23.2	25.4	61.1	58.2
y9	27	28.7	36.9	34.9	25.1	26.2	26.2	24.3	21.2	21.7	53.4	53.8
y10	22.9	22.5	25.3	25.4	17.3	21.2	21.7	20.4	15.3	16.6	48.4	54
y11	28.5	29.6	34.3	22	28.7	25.4	34.3	34.4	20.1	16.4	45.6	44
y12	38.9	34.9	41.1	41.7	37.1	37.4	37.1	36.6	26.7	25.3	59.1	54.4
y13	28.8	28.5	29.6	29.1	27	25.3	26.2	25.1	25.4	29	43.4	44.3
y14	27.4	30.8	32.2	31.9	29	29.8	31.6	28.7	19.8	18.1	48.9	43.1
y15	34.3	33.8	32.6	31.5	22.9	20.4	26	29.9	19.2	20.9	39.1	35.7
y16	34.3	34.7	35	33.2	21.1	18.7	32.9	32.1	22.1	19	43.7	43.3
y17	32.7	27.6	34.7	32.4	24.6	24.3	31.6	30.2	23.1	18	46.5	47.8
y18	34.1	29.6	26.5	31.8	24	23.7	38.3	35.2	21.7	22.8	38.4	38.6
y19	34.6	34.1	34.3	35.3	32.6	29.8	28.7	26.3	19.5	18.4	46.2	45.1
y20	26.8	25.3	23.7	22.1	27	24.6	23.9	23.2	17.8	15.3	46.7	42.5
y21	23.7	25.4	17.6	20.3	20.6	18	21.4	24	13.1	14.1	38.9	33.3
mean	31.3	30.64	32.67	31.7	27.55	26.4	30.74	30.3	20.98	20.7	48.31	47.1
SD	4.88	3.89	6.488	6.87	6.314	5.92	5.714	5.73	3.987	4.83	6.936	7.88
E7	23.5	23.7	19.8	22.6	16.1	16.4	24.8	24.5	12.8	15.6	37.2	35.8
E8	19.7	25.1	25.4	27.4	11.3	9.6	25.6	26	17.3	17.6	29.9	37.8
E9	29.6	29.6	33.3	31	20.6	27.1	24.3	22.9	16.1	15.9	48.4	43.9
E10	20.3	23.4	19.8	21.4	10	11.9	17.8	18.3	12.2	13.1	29.9	29.9
E11	25.1	28.2	27.1	25.3	20	23.1	30.2	28.4	29.4	35	15.2	15.5
E12	19.5	18.9	20.8	20.4	12.5	13.9	18.4	18.1	13.5	11.6	29.4	26.5
E13	24.8	26	15.5	15	10.8	10.5	18	20.4	11.9	13	27.4	24.5
E14	19.7	17.6	16.9	19.5	10.8	10	17.2	17	12.2	11.3	34.3	29.1
E15	20.1	20.1	19.4	19.2	20.1	17.3	16.7	17.6	15.9	14.9	28	27.4
E16	34.4	35.7	34.4	36.1	28	24.3	28.5	26.8	17.5	17.2	39.5	43.6
E17	24	27	18.9	19.4	18.6	17.8	21.2	19.4	15.2	13.8	30.7	32.9
E18	26.7	27.4	26.2	26.5	23.2	22.1	26.3	23.7	15	18.4	39.2	34.4
E19	30.1	29.6	21.7	25.7	22.1	22	16.1	18	17.5	20	32.4	31.8
E20	28.8	34.9	35.2	35.3	28.8	27	27.1	27.4	31	27.4	46.5	47.1
E21	27.9	29.3	28.8	25.9	18.6	19.7	25.3	24.6	12.7	16.1	33.9	30.4
E22	19.2	15.9	20.4	18.6	12.4	15.8	21.7	22.6	6.3	8	35.7	33.6
E23	30.1	27.6	25.4	24.8	22.5	21.1	27.3	28.2	16.1	15.9	47.2	44.8
E24	42.8	40.8	34.4	29	25.3	24.3	24	26.7	20.8	19.5	54.1	48.9
mean	25.9	26.71	24.63	24.6	18.43	18.6	22.81	22.8	16.3	16.9	35.49	34.3
SD	6.23	6.422	6.399	5.76	6.061	5.75	4.503	4.01	5.946	6.15	9.31	8.77
P-value	0.01	0.036	8E-4	0	1E-4	0	8E-5	0	0.01	0.05	6E-5	0

Left leg Lengths (cm)

Subject	Knee Extension	Hip Extension	Knee flexion	Hip Flexion	Ankle Dorsi-Flexion	Ankle Planar-Flexion
Y5	38	80	39	41	15	15
y6	38	83	41	42	15	15
y7	38	79	39	37	16	13
y8	39	80	39	40	16	13
y9	44	87	44	43	16	15
y10	37	77	37	42	13	12
y11	40	86	41	42	16	15
y12	44	90	45	47	14	16
y13	35	75	36	39	15	13
y14	36	76	37	40	15	14
y15	36	74	37	40	15	14
y16	40	86	41	44	17	15
y17	37	79	38	42	14	13
y18	44	91	44	46	15	15
y19	39	78	39	39	13	12
y20	39	78	38	40	14	14
y21	34	71	35	37	12	11
mean	38.70588	80.58824	39.41176	41.23529	14.76471	13.82353
SD	3.015889	5.712473	2.895229	2.750668	1.300452	1.380004
E7	38	78	37	40	14	13
E8	39	83	40	44	15	14
E9	38	78	38	37	15	14
E10	33	69	33	35	13	13
E11	46	87	44	45	13	17
E12	36	73	37	36	13	12
E13	38	80	39	39	15	14
E14	35	72	35	35	14	13
E15	38	81	39	39	13	13
E16	37	77	38	40	16	14
E17	38	85	41	44	15	14
E18	38	74	38	37	14	15
E19	40	84	40	45	15	13
E20	43	88	44	46	15	15
E21	40	80	40	41	16	15
E22	42	83	42	43	15	14
E23	37	79	39	38	14	14
E24	38	83	39	44	14	14
mean	38.55556	79.66667	39.05556	40.44444	14.38889	13.94444
SD	2.955	5.246848	2.754082	3.681787	0.978528	1.109967
P-value	0.882595	0.623088	0.711934	0.475337	0.343911	0.777839

Calculated MVIC Joint Torques (N-m) (larger of the two trials)

	Hip Ext		Hip Flex		Knee Ext		Knee Flex		Ank Dors		Ank Plntr	
Subject	R	L	R	L	R	L	R	L	R	L	R	L
Y5	352.29	350.45	144.65	128.17	145.61	134.06	156.32	159.38	39.827	37.632	82.47	89.67
y6	297.68	258.66	152.1	158.05	156.78	143.75	89.435	109.69	25.872	23.961	66.82	73.353
y7	298.07	320.52	141.61	142.86	141.78	126.99	114.7	113.9	37.338	46.726	78.75	73.892
y8	309.58	307.33	142.69	145.43	150.27	132.24	153.66	137.59	37.946	39.827	87.53	77.841
y9	314.87	314.61	112.94	110.41	115.56	123.75	107.88	112.97	28.518	34.026	70.66	79.086
y10	171.87	191.67	84.78	89.317	87.142	83.035	73.608	76.871	21.148	21.148	66.25	63.504
y11	270.05	289.08	164.34	141.59	148.18	116.03	108.54	115.32	35.907	31.517	71.59	67.032
y12	383.77	367.79	170.4	170.88	159.2	167.74	168.9	164.93	39.837	36.632	86.08	92.669
y13	243.55	217.56	112.09	100.14	108.66	98.784	97.02	95.256	36.77	42.63	69.15	56.438
y14	283.71	239.83	135	123.87	95.001	108.66	120.75	108.05	27.028	29.106	71.76	67.091
y15	221.95	236.42	114.28	117.21	89.004	121.01	105.87	83.035	28.538	30.723	54.47	53.645
y16	323.59	294.98	164.09	141.86	167.72	136.02	106.19	84.78	37.191	36.819	74.09	64.239
y17	285.77	268.65	139.53	130.07	103.15	118.57	104.72	91.61	31.007	31.693	59.24	60.897
y18	278.23	283.59	173.77	172.66	158.03	147.04	112.11	103.49	36.358	33.516	64.68	56.742
y19	285.89	269.83	120.1	109.69	135.63	132.24	129.57	124.6	25.607	24.843	65.62	54.331
y20	233.81	181.16	92.816	93.688	122.7	102.43	100.74	100.55	29.086	24.422	57.9	64.072
y21	157.25	141.25	88.837	87.024	97.412	84.633	55.037	70.658	17.052	16.582	42.15	41.934
mean	284.67	274.51	135.32	129.74	130.28	124.52	115.63	111.38	32.374	32.826	70.44	68.406
SD	50.979	52.767	27	25.589	27.218	20.553	25.308	25.304	5.9033	7.1385	9.639	11.871
E7	151.35	172.75	101.53	97.216	85.936	88.259	64.053	59.466	20.766	21.403	46.69	47.393
E8	224.73	222.87	109.56	112.11	93.472	95.932	69.943	44.296	35.133	25.872	48.98	51.862
E9	245.78	254.55	89.2	88.112	107.25	110.23	75.421	100.92	24.588	23.667	56.69	66.405
E10	133.29	144.71	57.31	62.769	61.152	75.676	44.953	38.485	15.543	16.689	41.66	38.093
E11	254.07	231.05	134.51	133.18	132.38	127.13	92.708	99.607	49.529	44.59	27.05	25.823
E12	145.04	148.8	63.504	64.915	70.707	68.796	46.922	50.401	17.199	17.199	39.51	34.574
E13	170.67	121.52	75.421	77.969	95.168	96.824	47.775	41.278	12.495	19.11	34.91	37.593
E14	121.62	137.59	64.827	58.996	77.969	67.571	40.817	37.044	16.601	16.738	41.02	43.698
E15	152.52	154	77.969	67.267	93.639	74.852	67.267	76.822	18.228	20.257	37.51	35.672
E16	261.68	272.41	98.99	111.72	136.89	129.45	101.25	104.27	29.361	27.44	57.9	59.819
E17	183.26	161.6	98.608	91.414	99.176	100.55	67.091	74.735	25.431	22.344	50.57	45.139
E18	215.56	192.18	89.2	95.364	102.98	102.04	86.024	86.397	26.617	25.245	55.7	57.624
E19	198.06	181.93	81.928	79.38	122.69	117.99	90.807	86.24	29.4	47.628	56.45	41.278
E20	314.61	304.43	115.98	123.52	143.44	147.07	125.13	124.19	32.046	45.57	63.21	69.237
E21	205.16	225.79	100.52	101.66	107.41	114.86	78.792	77.224	25.284	25.245	51.45	49.833
E22	176.79	165.93	103.23	95.236	105.27	79.027	60.505	65.033	17.787	11.76	54.61	48.98
E23	204.08	196.65	110.23	105.02	106.24	109.14	83.045	85.995	24.01	22.089	57.08	64.758
E24	267.39	279.81	118.11	115.13	164.97	159.39	108.54	96.697	33.751	28.538	67.91	74.225
mean	201.43	198.25	93.924	93.387	105.93	103.6	75.058	74.95	25.209	25.632	49.38	49.556
SD	52.738	53.806	20.811	21.633	26.279	26.238	23.225	25.678	8.9614	10.267	10.64	13.436
P-value	0.0003	0.0013	8E-05	0.0003	0.0193	0.03	0.0002	0.0005	0.0261	0.0524	1E-5	0.0005

Composite Scores using Principal Components

Subject	Composite Strength	Composite ROM
y5	5.792041	3.53416
y6	2.372851	-0.74963
y7	4.330234	2.85286
y8	6.058161	4.11503
y9	4.099838	3.76745
y10	-4.1352	1.10481
y11	1.491825	4.45083
y12	3.997446	1.94616
y13	5.491003	1.74838
y14	6.756263	4.13993
y15	-2.7781	3.336806
y16	6.915837	1.01877
y17	5.807012	1.41834
y18	-3.1955	0.04602
y19	4.728207	-0.43671
y20	5.787967	0.88093
y21	-1.3552	-0.96111
mean	3.068511	1.894884
SD	3.70512	1.809153
E7	-7.50905	0.997026
E8	-2.72698	0.728987
E9	-1.35012	-6.48546
E10	-4.90992	-3.77848
E11	-4.68008	-2.37104
E12	-7.25395	1.610678
E13	-2.78136	0.23274
E14	-5.57335	1.61836
E15	-6.10377	1.215311
E16	-5.85377	-5.96182
E17	-5.10031	-1.01113
E18	-1.95102	-4.36104
E19	-3.92956	-4.65681
E20	2.18083	-1.94622
E21	-6.18394	-1.91409
E22	-5.5193	-6.2514
E23	-3.7397	-0.86858
E24	-4.22353	-3.32272
mean	-4.28938	-2.0292
SD	2.351	2.783443
P-value	1.8E-07	2.66E-05

APPENDIX D. COMPARISON OF LOCAL DYNAMIC STABILITY FIT METHODS

Two methods have been developed to describe the local divergence behavior in gait. The maximum finite-time Lyapunov exponent method has been chosen for this dissertation over the double-exponential method, for the ease of interpretation. Both methods give the same results, but the double-exponential results can be difficult to interpret given this data. The double-exponential method is shown here.

Mean Divergence

The local stability of a system can be described by the response of that system to small perturbations. When a perturbation is introduced to the system moving through its state space, the system will be "bumped" to a nearby part of the state space. The system's new trajectory may converge back to the original trajectory, parallel it, or diverge away. The *rate* of divergence of neighboring state trajectories estimates local dynamic stability by measuring, on average, how quickly the system would diverge away from the original trajectory after being "bumped" over to a neighboring trajectory (Figure 2-4C). This divergence can be measured from the successive Euclidian distances between the points on the original and the "perturbed" trajectories (Rosenstein, Collins et al. 1993). Mean divergence quantifies the average rate of divergence between neighboring trajectories in state space. A steeper increase of mean divergence indicates that the system diverges faster from its original path in response to a small perturbation, and thus exhibits greater local dynamic *in*stability.

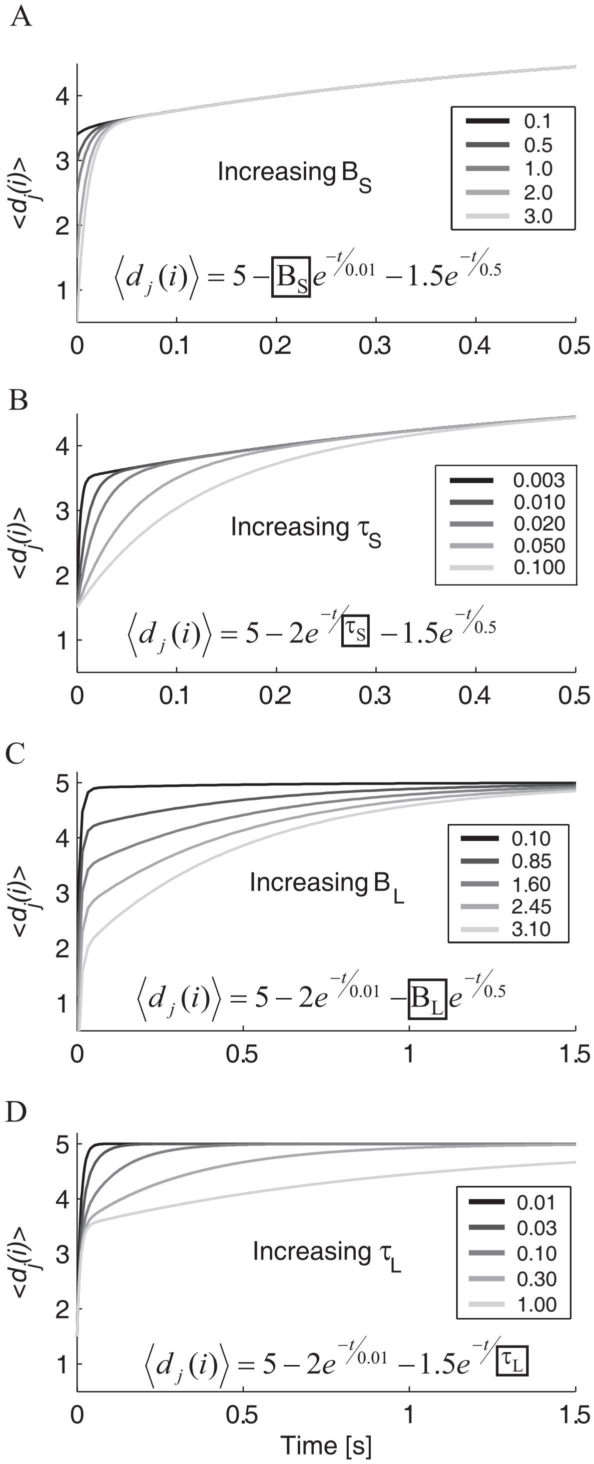
Mean divergence of the nearest neighbor trajectories was calculated using a previously published algorithm (Rosenstein, Collins et al. 1993) that was modified to use the state space trajectories defined above rather than delay-reconstructed state spaces from a single time-series. For each point $S(t)$ on the state-space trajectory, the nearest neighboring point $S(t^*)$ on an adjacent trajectory (excluding points on the same trajectory) was determined, forming the j^{th} pair of nearest neighbors. Euclidean distances between each pair of subsequent points on the two trajectories were then calculated. For this j^{th} nearest neighbor pair of $S(t)$ and $S(t^*)$, this formed a vector of Euclidean distances $d_j(i)$:

$$d_j(i) = \left\| S(t + i\Delta t) - S(t^* + i\Delta t) \right\|_2 \qquad (3\text{-}18)$$

and $d_j(i)$ is the Euclidean distance between the each pair of points after each discrete time step i (i.e. $i\Delta$t s) on the two trajectories. The local divergence was computed out to 10 seconds beyond the initial perturbation. This process was repeated for all initially neighboring points from the data set and the $d_j(i)$ for each pair of points were averaged to define the mean divergence vector, $\langle d_j(i) \rangle$, where $\langle \cdot \rangle$ denotes the arithmetic mean over all values of j. A double-exponential function was used to parameterize these divergence curves (Kang and Dingwell 2006):

$$\langle d_j(i) \rangle = A - B_S e^{-t/\tau_S} - B_L e^{-t/\tau_L} \qquad (3\text{-}19)$$

where τ_S and τ_L ($\tau_L >> \tau_S$) represent the time constants that describe how quickly $\langle d_j(i) \rangle$ saturates to A, and B_S and B_L determine the size of the effect the dynamics at each timescale have on $\langle d_j(i) \rangle$ (Figure 3-2).



Sensitivity of Divergence Curves.

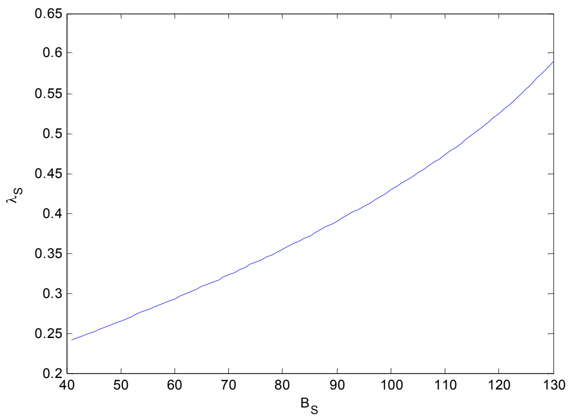
Changes in the shape of the double exponential fit curves as functions changes in each of the different parameters given in Equation 3-19. As each of the parameters noted increases in value, the lines get lighter and shift to the right. Within each subplot, all parameters other than the parameter being varied were fixed at a nominal value (i.e. Plot A shows effects of changing parameter B_S while keeping others constant). Note that the horizontal scales in subplots A and B are different than those used in C and D to show greater detail.

To demonstrate the differences in the two methods, the relationship of the two sets of measures are given here. A double-exponential curve was defined, and then the slopes of its natural logs were calculated.

Part 1. Vary B_S

These parmeters were fixed: A = 400; τ_S = .05; B_L = 250; τ_L = 10;

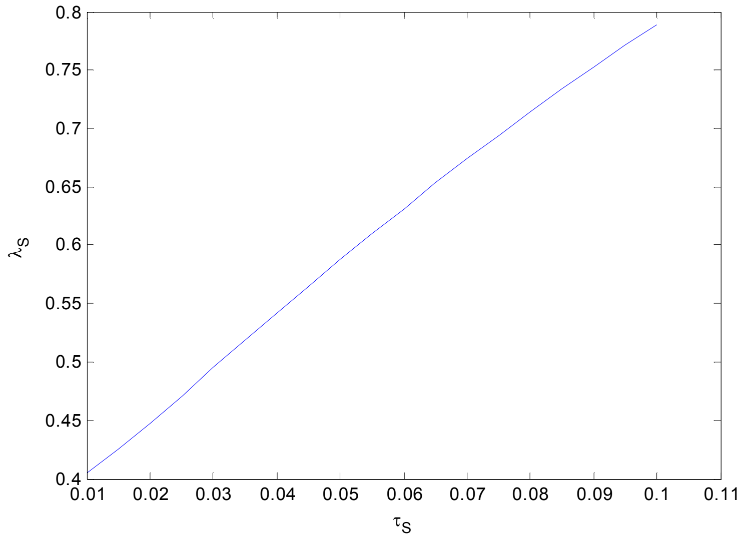
BS varied between 41 and 130.



Part 2. Vary τ_S

These parameters were fixed. A = 400; B_S = 75; B_L = 300; τ_L = 10;

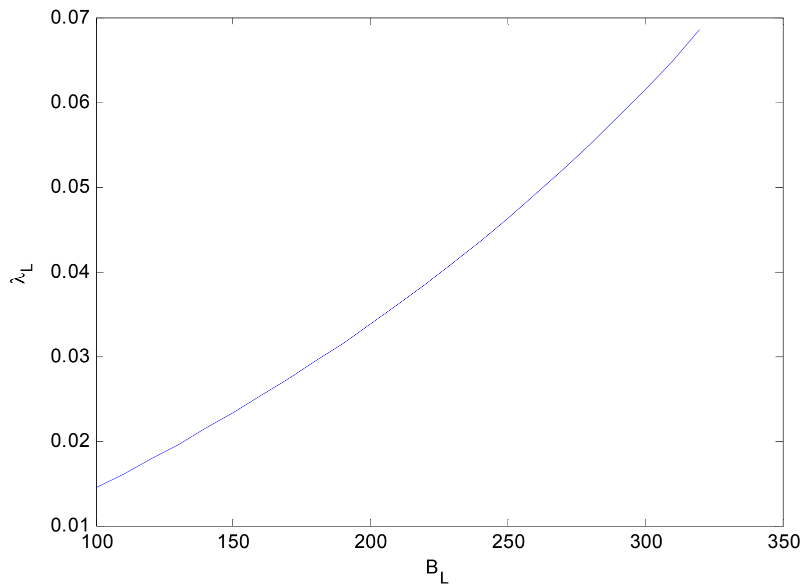
TS= was varied between .01 and .1.



Part 3: Vary B_L

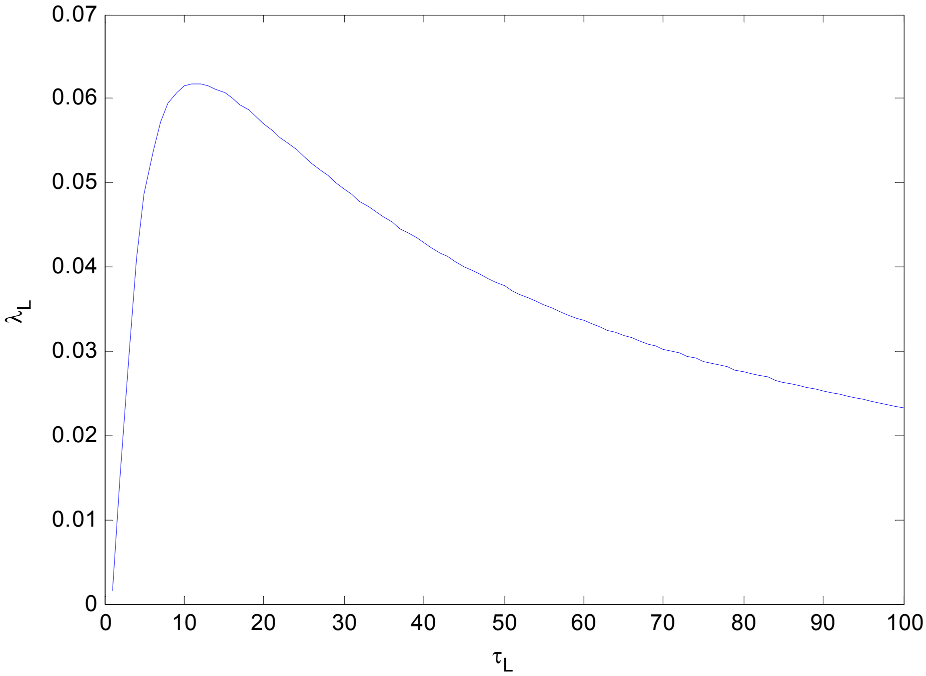
Parameters fixed: A = 400; $B_S = 75$; $\tau_S = .05$; $\tau_L = 10$;

BL varied between 100 and 324;



Part 4: Vary TL
Fixed Parameters: A = 400; $B_S = 75$; $\tau_S = .05$; $B_L = 300$;

TL varied between 1 and 100;



For τ_L, the range of interest is 5-20, which is right around the "hump." Because of this, τ_L cannot be used to understand ll in the context of this dissertation.

λ_S and τ_S have a near-linear direct relationship, and a same interpretations can be made using either measures, but not for the long-term measures.

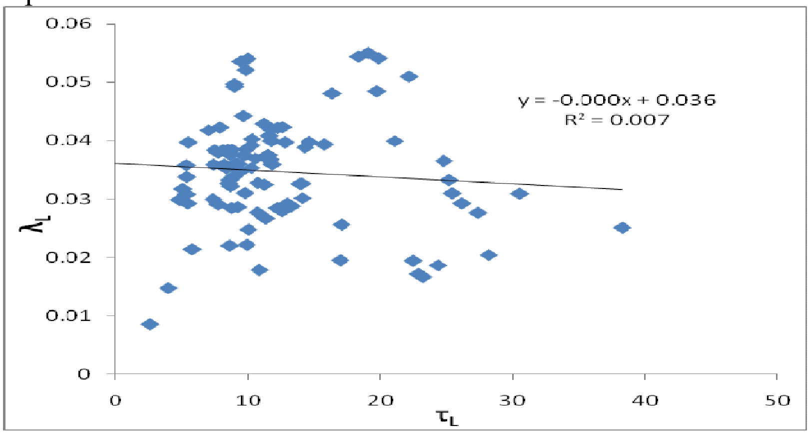
Comparison of Data

Data from a previous study (Kang and Dingwell 2006) was used. Data was collected from twenty healthy adults age 18-75. Trunk motion was measured as subjects walked for 5 minutes at their preferred walking speed. A state-space was defined as shown:

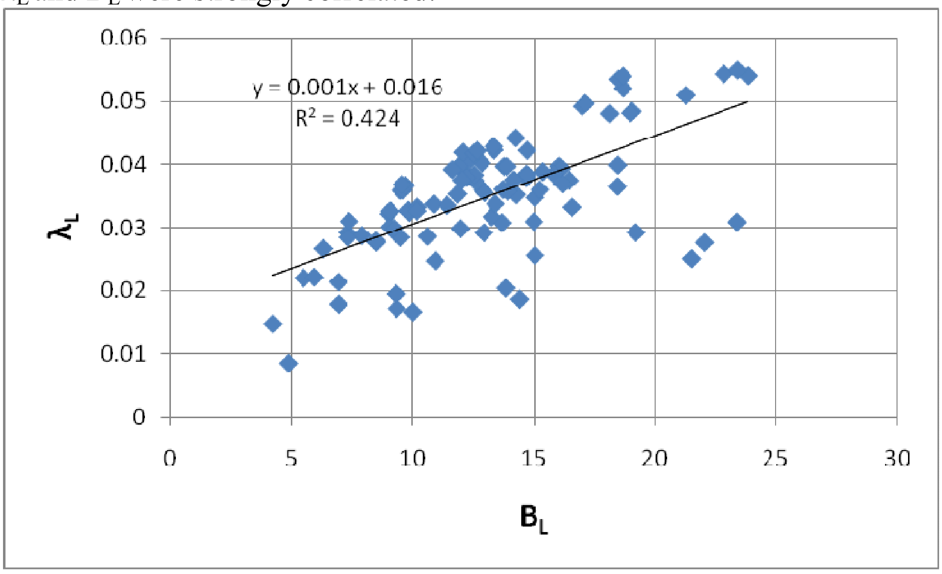
$$S(t) = [x, y, z, \dot{x}, \dot{y}, \dot{z}, \theta, \phi, \psi, \dot{\theta}, \dot{\phi}, \dot{\psi}]$$

Mean log divergence (MLD) and mean divergences (MD) were calculated from the first 1, 2, 3, 4, and 5 minutes of the data trials. From MLD curves, their slopes between 0-1stride, λ_S, and 4-10 strides λ_L values were calculated. From MD curves, a double exponential fit was performed as described in Chapter 5.

As described in Chapter 5, λ_L and τ_L were not strongly correlated, as expected from our previous simulation results.

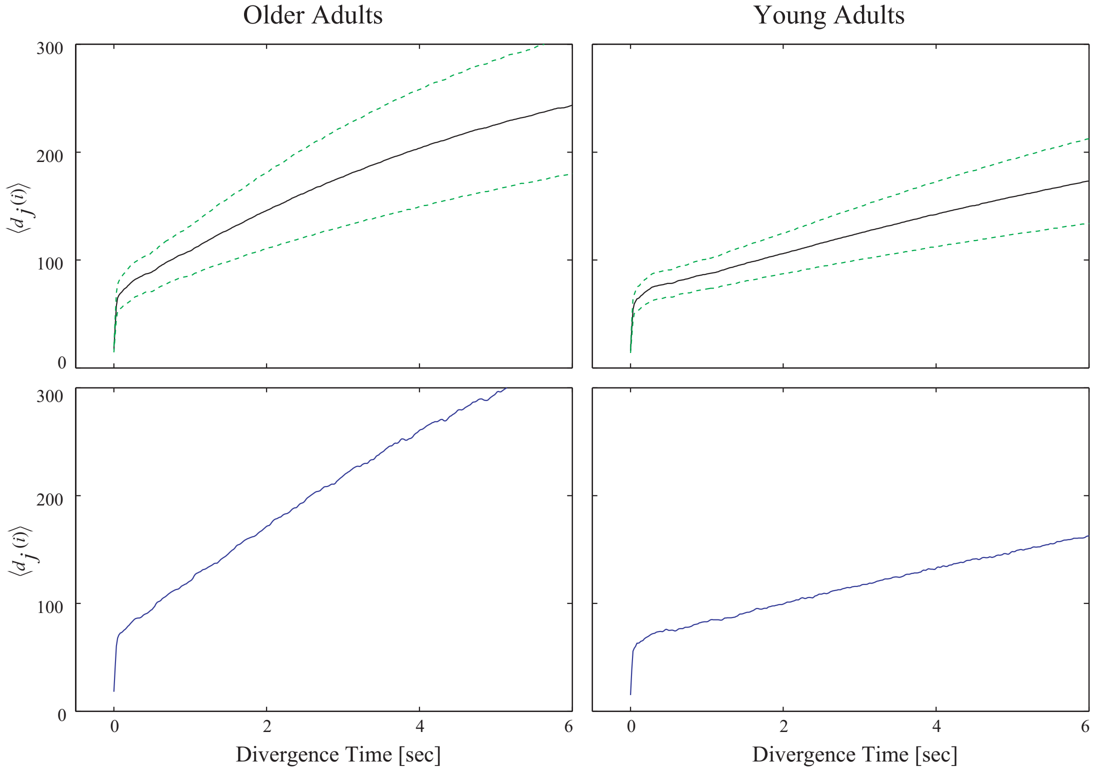


λ_L and B_L were strongly correlated:



Using the double-exponential method, the following results can be obtained.

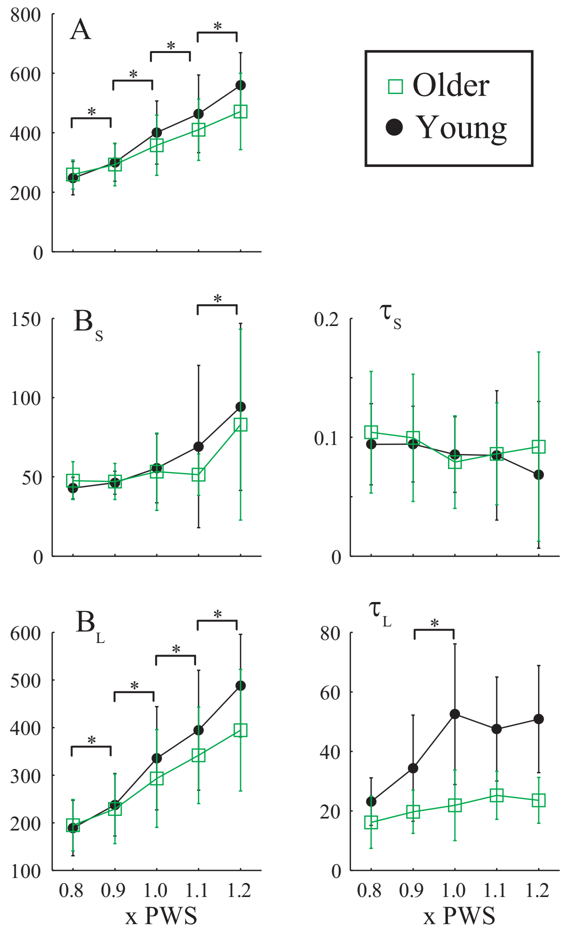
From Aim 2:



Sample Mean Divergence Curves.
Divergence curves are displayed for the 1x PWS speed. Top: Group averages with standard deviation bars (dotted line). Bottom: A typical trial from each group (left: older adult, right: young adult). Older adults displayed greater between-subjects variability compared to young adults, and larger divergence values, even though their walking speeds were not different.

Mean Divergence curves are shown in Figure 5-1. A, B_S and B_L increased with speed ($p < 0.001$), but did not differ significantly between age groups. τ_S did not vary significantly with age or speed. A, B_S and B_L increased with walking speed, reflecting the larger velocities and accelerations experienced during faster walking speeds. τ_L for 0.8x and 0.9x speeds were significantly different from the other three speeds. τ_L was smaller in older adults, reflecting that small deviations lead to different kinematic patterns at a much faster rate in older adults. This indicated that older adults exhibited higher sensitivity to perturbations.

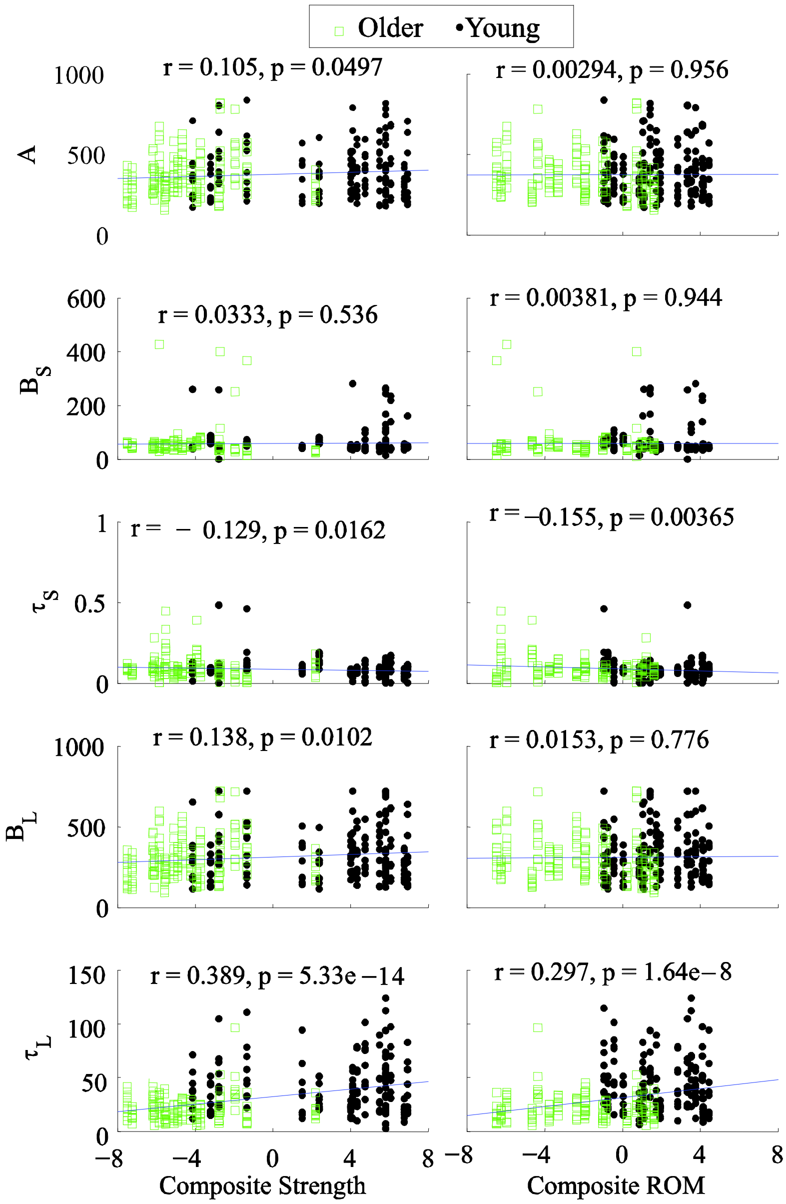
There was a significant interaction of age and speed for A, and B_L where age-differences were more noticeable at higher speeds ($p < 0.0006$), and for τ_L, where the effect of walking speed was more pronounced in young adults ($p < 0.001$; Figure 5-2). When either Strength or ROM composite score was included as a covariate, age and speed effects on τ_L were still significant ($p = 0.0052$). Age-effect was no longer significant if both covariates were included ($p \geq 0.011$; Figure 5-3), suggesting that strength and range of motion help explain the the age-related differences in local dynamic stability.



Double-exponential Fits vs. Speed. Error bars denote standard deviations within each group. Horizontal brackets denote significant Tukey's LSD post-hoc comparisons at $p < 0.005$.

P-values for Local dynamic stability

	A	B_S	τ_S	B_L	τ_L
Age	0.218	0.398	0.618	0.195	8.853E-06
Speed	3.896E-70	6.809E-08	0.055	4.64E-68	6.57E-17
Age x Speed	0.000578	0.585	0.446	0.000517	1.071E-06



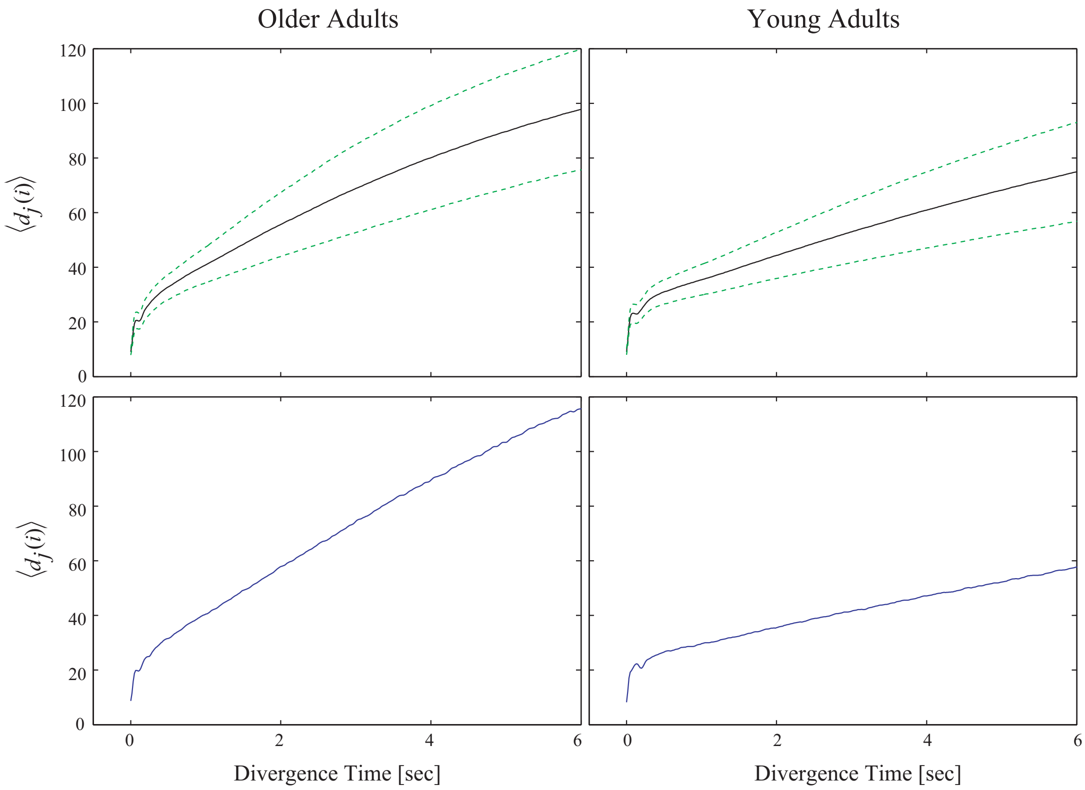
Relationship of mean divergence fit parameters to Strength and ROM. Results from all speeds are shown.

From Aim 3:

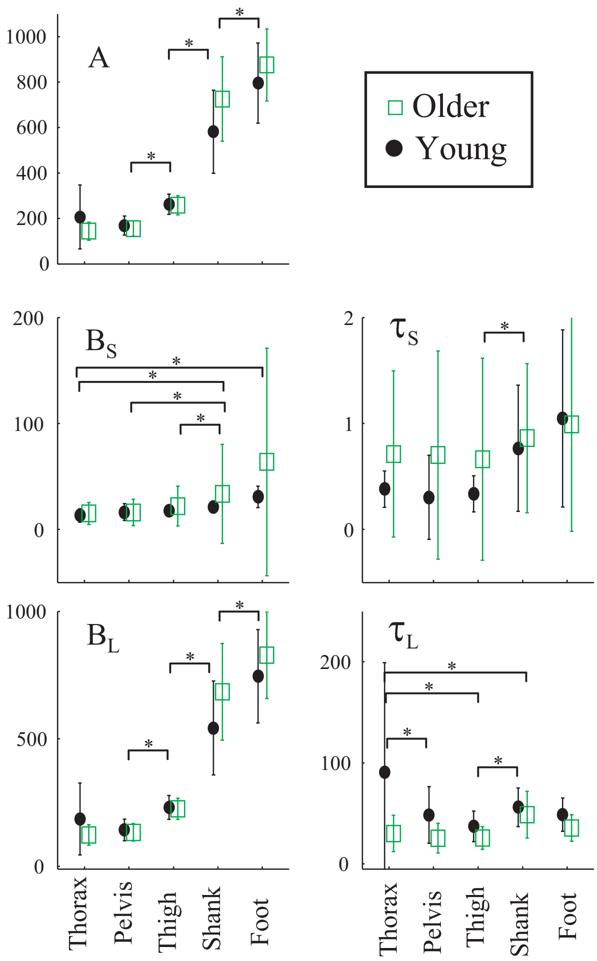
Local Divergence

Sample mean divergence curves are shown in Figure 6-2. A, B_S, τ_L and B_L were higher in lower segments. ($p < 0.001$; Figure 6-3). Only τ_L was significantly higher in young adults, and also showed a significant interaction of age and segment ($p < 0.001$), where the age-related differences were more pronounced in superior segments (Figure 6-3). Segment effect was significant ($p < 0.001$), but there was no pattern related to segment height. The actual values for the fit parameters in the trunk are not the same as those in Aim 2, because different state variables of different sizes were used. However, the trends are consistent.

Mean Divergence fit parameters A, B_S and B_L were larger in inferior segments, reflecting the larger motion of inferior segments, and corresponding increase of divergence between nearby trajectories. We are also interested in the *rate* of divergence in response to perturbations as a measure of stability. There was overall trend for superior segments to exhibit larger τ_L, but τ_L was larger in the shank compared to the pelvis or the thigh. The higher τ_L values in young adults indicate that given a perturbation, they would diverge away much slower, and thus are more resistant to perturbations.



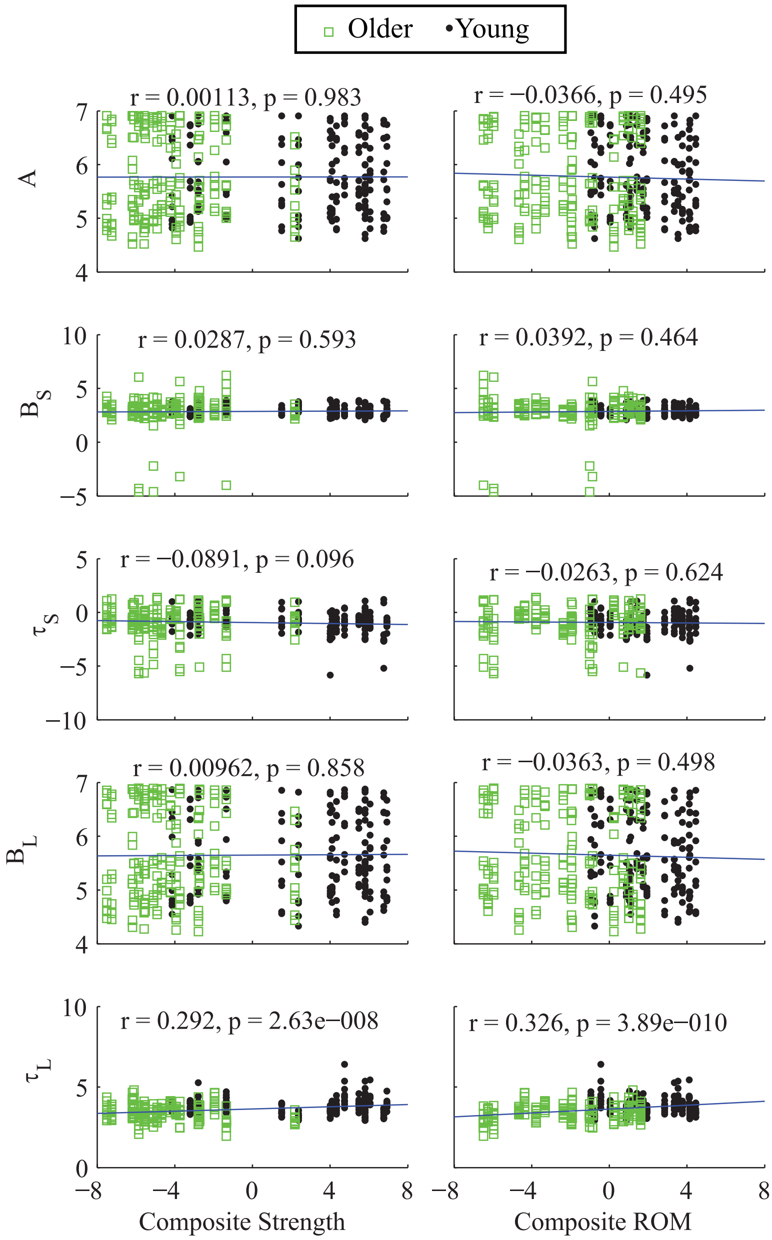
Mean Divergence Curves of the Pelvis Segment
Mean divergence curves are displayed for the pelvis. Top: Group averages with standard deviation bars (dotted line). Bottom: A typical trial from each group (left: older adult, right: young adult). Older adults display a larger divergence curves.



Interaction Plot of Age and Segments for Mean Divergence Fit Values. Error bars denote standard deviations within each group. Horizontal brackets denote significant Tukey's LSD post-hoc comparisons at $p < 0.005$.

P-values for Local Dynamic Stability Comparisons

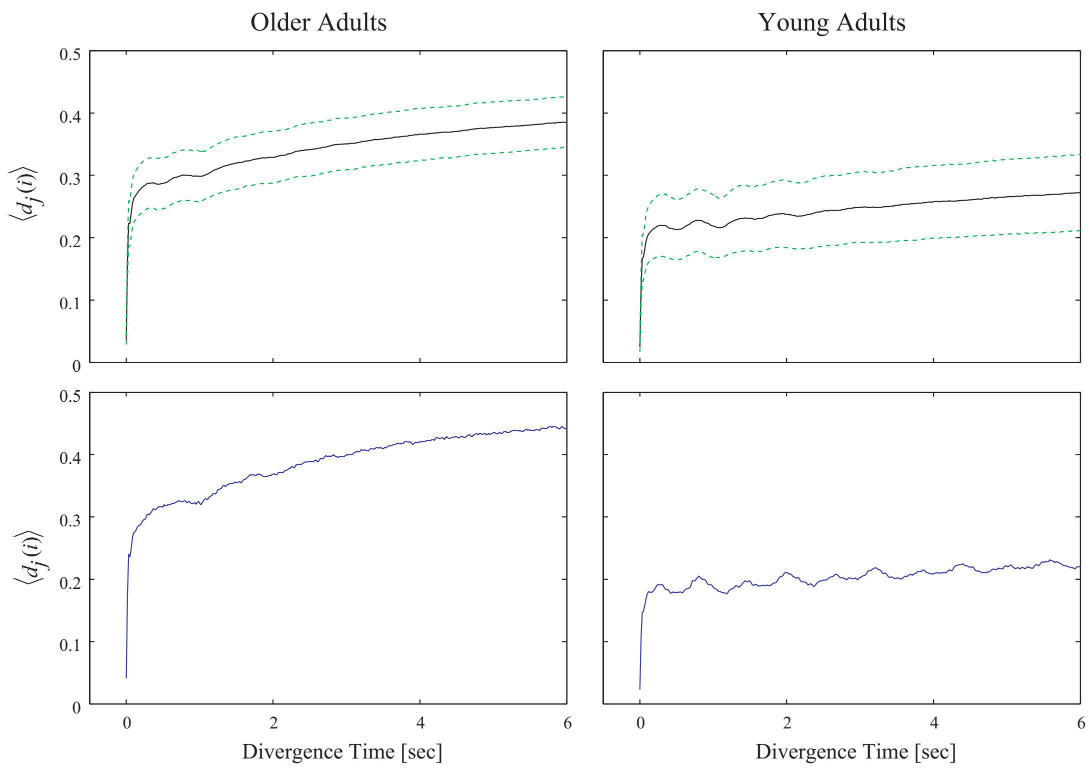
	A	B_S	τ_S	B_L	τ_L
Age	0.110	0.187	0.733	0.212	0.02087
Segment	1.24E-146	1.86E-90	1.018E-06	6.169E-126	7.848E-21
Age x Segment	4.76E-08	0.000996	0.5406	4.81E-07	4.793E-15



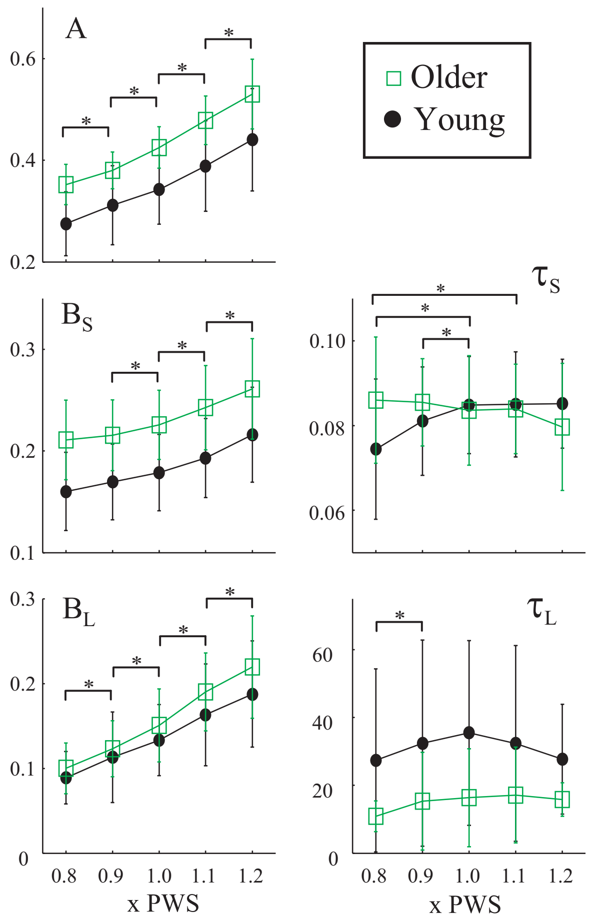
Relationship of divergence fit values to Strength and ROM
Log-transformed fit values are shown.

Aim 4:

All fit parameters A, B_S, τ_S, B_L and τ_L were sensitive to speed ($p < 0.0055$), while τ_L was greater in younger adults ($p < 0.004$). Parameter A trended toward being larger in older adults ($p < 0.03$). Significant age x speed interaction was found in τ_S and τ_L ($p < 0.001$), where differences in τ_S between groups were more pronounced at lower speeds (pairwise comparison, $p < 0.003$). Speed effects on τ_L were more pronounced in older adults (Figure 7-6).



Mean Divergence Curves
Mean divergence curves are displayed for the 1x PWS speed. Top: Group averages with standard deviation bars (dotted line). Bottom: A typical trial from each group (left: older adult, right: young adult). Older adults displayed larger size divergences even though the walking speeds were not different between groups.



Divergence Fit values vs. Speed. Speed effects were noticeable in A, B_S and B_L. τ_L was larger in young adults. Error bars denote standard deviations within each group. Horizontal brackets denote significant Tukey's LSD post-hoc comparisons at $p < 0.005$.

P-values for local dynamic stability comparisons

	A	B_S (log)	τ_S (log)	B_L(log)	τ_L(log)
Age	0.0386	0.420	0.361	0.390	0.00260
Speed	1.246E-65	7.029E-43	0.00421	2.520E-58	0.000247
Age x Speed	0.917	0.2707	1.600E-07	0.988	0.590

APPENDIX E. CALCULATION OF POINCARÉ SECTIONS AND SUBSEQUENT METRICS

Because data was sampled in discrete timesteps, the calculation of the Poincaré section, and its intersection with the stride trajectories in state-space, at each percentage of the gait cycle requires special considerations. The MATLAB code is shown in Appendix F.

First, a state-space was defined using the appropriate state variables. Each variable is really a vector of numbers as a function of discrete time.

Second, each variable was time-normalized based on gait events. From here, an average stride was defined, as a function of % gait cycle. From the time-normalization information, each data point was re-defined as a function of the % gait cycle. Their position in state-space does not change, since their actual values stay the same.

Third, Poincaré section was defined as a hyperplane that is normal to the average trajectory for each % gait cycle. The tangent vector to the trajectory at n% of the gait cycle was determined numerically as the vector that links the previous point (n-1)% and (n+1)% of the gait cycle.

Fourth, the data points near the Poincaré section were identified. For n% of the gait cycle, any data points that were defined within ±5% of n% were found. Then, for each data point identified, an algorithm tested whether points temporally subsequent to the data point are heading toward, or away from the Poincaré section. Any points on the same stride subsequent to each other, and thus redundant for finding the intersection of the stride trajectory and the Poincaré section, were removed. By following the trajectory, a data point (P-) was identified for each stride, that was just before crossing the Poincaré section. That is, points whose subsequent point (P+) was on the other side of the Poincaré section were identified. P- points were checked to see if they occur on regular intervals. Sometimes P- points occurred irregularly, because that stride behaved differently from others. Such points were discarded from further analysis, but this fact was tracked for later use. Particular to Chapter 7, if a part of the data was unusable due to noise, etc, these sections were "masked" based on the "noise vector" defined in Chapter 7. Any P- points that fell into this masked region were also discarded.

Fifth, a line was defined between P- and P+ points were defined, and the point of intersection between this line and the Poincaré section was calculated for each stride. These intersection points defined the cross-section of the state-space at that % gait cycle. Any strides associated with a discarded P- were not used in the calculations.

For orbital stability calculations, the vector between intersections S_k, as defined in Chapter 3, and the S^* from the average cycle at that % gait cycle, were calculated. Since Floquet Multiplier calculation uses consecutive strides, if stride k was discarded for this % gait cycle, then the mapping of stride k-1 to k, and k to k+1 were eliminated from the calculation of the Jacobian matrix. Thus the data matrices used in Eqn 3-17 would map k-3 to k-2, k-2 to k-1, then skip to k+1 to k+2, etc. This made use of all available data.

For variability calculations in state-space as used in Chapter 6, the RMS distance of the intersection points to S^* was calculated.

APPENDIX F. MATLAB CODES.

rosenmld.m is an implementation of Rosenstein (1994) algorithm to calculate divergence curves.

Rosenemg.m is a modification that masks out data flagged by the noise vector.

Lyapfit.m calculates the fit parameters from the divergence curves

Sosbmax.m calculates the Poincaré sections and Floquet multipliers

Sosbmaxemg.m is a modification that masks out data flagged by the noise vector.

sosbmax.m

```
function [EFM, Var, DRMS, Poincare] = sosbmax(DATA, LHS, GCC)

   EFM=0;
   AlphaTS=0;
   AlphaRT=0;
   Var=0;
   EFM=0;
   MPF=0;
   SDT=0;
   devDATA=0;


      %%calculate DRMS
   [m,n]=size(DATA);
   DATAa = DATA - repmat(mean(DATA,1), m ,1);

   DRMS = (mean(sum(DATAa.^2,2)))^.5;


format compact
%% sosbstab.m
%% calculates stochastic orbital stabilty, instantaneous FM, and
%% Variability in a stochastic van der Pol oscillator
%% MArch 12, 2007, Hyun Gu Kang
%% Last modified:  May 7, 2007

%% taken in n-dimensional state space matrix
%% inputs:  DATA = matrix of data, each column = state variable, and each
%% row is time point
%%          LHS = Left heel strike data (or other demarkations of cycles
%%          GCC = the gait cycle parts of interest: i.e, 1:100 for all, or
%%          30:40 if you only want to look at a particular % gait cycle.

%%  Dependent Measures

EFM = zeros(100,1);

%downsample
[m,n]=size(DATA);
faze = zeros(m,1);



for S=1:n %for each state variable

   %% Finite fourier series
```

```
    xo = mean(DATA(4001:end,S));  %get the center


    % Normalize
    NumStrides = length(LHS)-1;
    Strides=zeros(NumStrides-1,101);

    for i = 1:NumStrides-1
        BeginStrideSamp = LHS(i);
        EndStrideSamp = LHS(i+1);

        if EndStrideSamp>m
            break;
        end
        [NormStride,time] = normalfuLCM(DATA(BeginStrideSamp:EndStrideSamp,S));
        %[FineStride,time] = normalfuLCM(DATA(BeginStrideSamp:EndStrideSamp,S),1e3);
        Strides(i,1:101) = NormStride;
        %FineStrides(i,1:1e3) = FineStride;
        faze(BeginStrideSamp:EndStrideSamp) = linspace(0,100,EndStrideSamp-BeginStrideSamp+1);

    end %-- for i = 1:NumStrides


    Str = reshape(Strides', 1, numel(Strides));
      Amaaa = mean(Strides,1)';%[ xref(99) xref ]; %size = 102 [ 99% 0% .... 99% 100%]

    Amean(:,S)= [Amaaa(95:100,1); Amaaa(:,1); Amaaa(2:7,1)];

end % S


%figure(1)


% for defining the section, use 99%- 0/100% - 2 % .... 99%, 100%.  Sections
% defined only at 0-99%.  100% assumed to be equal to 0%

%define Poincare section as a line perpendicular to the points, finds slope
%of things crossing the line and plots them as proposed originally
K = [1,2,5,10,20,30,40];  FM = zeros(100,1);
clear S

TS=1;
AlphaTS=0;

if TS==1

    for gc= GCC+1       %gc = 2 really is 0%, so we study 0%-99%
        Current_gc = gc-1
```

```
figure(1)
%subplot(2,2,1)
cla
%plot3(DATA(:,1),DATA(:,2),DATA(:,3),'c','LineWidth',0.5)
hold on
%plot3(Amean(:,1),Amean(:,2),Amean(:,3),'r.-','LineWidth',3)
hold on

%step 1: determine flow vector, T in the document
%T = (amean((gc-1)*100+1,:)) -amean((gc-1)*100-1,:);
T = Amean(gc+1,:)-Amean(gc-1,:);

% step 2:  define S*n, current fixed point
Sn = Amean(gc,:);

% Method 1:  find points right before and right after the Poincare section

%1. find cluster of points nearby the "fixed point"
[m,n]=size(DATA);
%minpt = zeros(7000,101);


dist=sum((DATA-repmat(Sn,m,1)).^2,2);  %calculate (Euclidian distance)^2 between fixed point and
all points in the data
Youter = dist<100^2*3;  % 0.05 is adequate for this simulation of this stochastic van der Pol
sumYo = sum(Youter);
Yinner = dist<200^2*3;
%Use phase information instead:
%A1 = r% before the current GC
%A2 = r% after the current GC
r = 5;
A1= gc-1-r-2;
A2= gc-1+r;

if A1 <0
   Y1 = faze>(A1+100);
   Y2 = faze<A2;
   Y = Y1+Y2;

elseif A2 >100
   Y1 = faze>(A1);
   Y2 = faze<(A2-100);
   Y = Y1+Y2;
else

   Y1 = faze>(A1);
   Y2 = faze<(A2);
   Y = Y1.*Y2;
end

Y=Y.*Youter;%+Yinner;
sumY=sum(Y);
```

```
%subplot(2,2,2)

% ylim([0.5 1.5])
hold on
%subplot(2,2,1)
a=0;
clear NN
for j = 1:m
   if Y(j) >0
      a=a+1;
      NN(a)=j;
   end
end
clear a
a=0;
clear NN2
for j = 1:m
   if Yinner(j) >0
      a=a+1;
      NN2(a)=j;
   end
end
clear a

NN = [NN-2, NN, NN-1];
%NN=[NN-10, NN-9, NN-8, NN-7, NN-6, NN-5, NN-4, NN-3, NN-2, NN-1, NN, NN+1, NN+2,
NN+3, NN+4, NN+5, NN+6, NN+7, NN+8, NN+9, NN+10, NN+11];
NN=NN.*(NN>0).*(NN<=m);
NN=sort(NN);
NN=deldup(NN);

NNlength = length(NN);
[M]=length(NN);
%NN = sample#'s of points nearby the fixed point
kay = 5;


Z=zeros(m,1);
%find points in NN2 that are not in NN
a=0;
clear NN3
for o=1:length(NN2)
   if (sum((NN2(o))==NN))==0
      a=a+1;
      NN3(a) =NN2(o);
      Z(NN2(o)) = 1;

   end
end
```

```
clear a

minpt_raw=zeros(M,1);

for j = 1:M %for each point in NN
   hold on

   %
   clear D Br cost D2
   D=0; cost=0; D2=0;

   for k = 1:kay
      %figure(2)
      if NN(j)+k-1 > m
         break
      end
      Sk =DATA( NN(j)+k-1,:);
      VPD = Sk - Sn;  %vector from P to data
      cost(k) = dot (T,VPD) /norm(T)/norm(VPD);
      D(k) = dstptpln(Amean(gc,:),T, DATA(NN(j)+k-1,:)); %track distance as it changes while we
follow the trajectory
      D2(k)= norm(Sn-DATA(NN(j)+k-1,:));
   end

   [y,I]=max(cost>0);

   [y,I2] = min(D);
   [y,I3]= min(D2);

   if abs(I-I3) > 4
      break
   end


   if I>1 && I<(length(cost)) && sum(NN(j)+I-2 == NN )

      minpt_raw(j) = NN(j)+I-1-1;
   end

end % j


% get rid of duplicates

minpt  =deldup (minpt_raw);
minpt = sort(minpt);

l = length(minpt);

%fill in "missing" minpt and eliminate minpt too close to each other
```

```
ADM = round(mean(diff(LHS)));
DM = diff(minpt);

while max(DM)>1.6*ADM
   for v=1:length(minpt)-1
      if DM(v) >1.6*ADM
         minpt=[minpt;minpt(v)+ADM];
      elseif DM(v) <.5*ADM
         minpt(v)=0;
      end
   end
   minpt  =deldup (minpt);
   minpt = sort(minpt);
   DM=diff(minpt);
end

for v=1:length(DM)
   if DM(v) <.8*ADM
      minpt(v)=round(minpt(v)+.2*ADM);
   end
end

for v=1:length(minpt)-1
   if DM(v) <10
      minpt(v+1)=0;
   end
end

if max(minpt) > m
   [wai,ai]=max(minpt);
   minpt(ai) =0;
end


minpt  =deldup (minpt);
minpt = sort(minpt);
l = length(minpt)

q=0;
%check minpt to see that it is in fact right before the plane
clear cost costp
for j=1:l

   oldpt=minpt(j);

   VPD = DATA( minpt(j),:) - [Amean(gc,:)]; %vector from P to data
   cost(j) = dot (T,VPD) /norm(T)/norm(VPD);

   while cost(j)>0

      minpt(j)=minpt(j) -1;
```

```
        %plot3(DATA(minpt(j),1),DATA(minpt(j),2), DATA(minpt(j),3),'m*')



        VPD = DATA( minpt(j),:) - [Amean(gc,:)];  %vector from P to data
        cost(j) = dot (T,VPD) /norm(T)/norm(VPD);
        q=q+1;
    end

    %drawnow


    if (minpt(j) <m)

        VPD = DATA( minpt(j)+1,:) - [Amean(gc,:)]; %vector from P to data
        costp(j) = dot (T,VPD) /norm(T)/norm(VPD);

        while (costp(j) <0) && (minpt(j)<(m-1))
            minpt(j) = minpt(j) +1;

            %plot3(DATA(minpt(j),1),DATA(minpt(j),2), DATA(minpt(j),3),'m*')

            VPD = DATA( minpt(j)+1,:) - [Amean(gc,:)];   %vector from P to data
            costp(j) = dot (T,VPD) /norm(T)/norm(VPD);
            q=q+1;
        end

        %drawnow


    end





    if abs(minpt(j)-oldpt)>15
        minpt(j)= inf;
    end
    q=0;

end



prept = minpt;
l=length(prept)

%%now we have all the points right before it crosses the poincare
%%section
```

```
%% skips
a=0;
clear skips
for j=1:l
   if prept(j)==inf
      a=a+1;
      skips(a)=j;
   end
end



%% Calculate dependent measures

clear Ixs x costhet DD Sigma FM

for j=1:l %for each cycle

   if (prept(j) ~= inf)

      %find intersection of trajectory and Poincare section
      %plane is defined by T dot (x-S*)=0,
      %line is defined as x = P1+u(P2-P1)

      uu = dot(T, Sn-DATA(prept(j),:))/dot(T,DATA(prept(j)+1,:)-DATA(prept(j),:));
      Ix = DATA(prept(j),:)+uu*(DATA(prept(j)+1,:)-DATA(prept(j),:));

      Ixs(j,:) = Ix;    % so Ixs is the state exactly at a % gait cycle

      DD(j,:) = Ix-[Amean(gc,:)]; % D vector in proposal
      xkx(j) = norm (DD(j,:));

   end

end %j

% store intersections in a STRUCT

Poincare(gc-1).ix = Ixs;
Poincare(gc-1).FP = Amean(gc,:);


%variability
Var(gc-1) = std(deldup(xkx));


%Extended Floquet Multiplier

for dela = 1:1
```

```
        delay = (dela);  %extended Floquet Multiplier
        %need to account for non-consecutive data:
        RQ = Ixs;

        [m,n]=size(RQ);
        Fpt = Amean(gc,:);   %this is S*
        RQ=RQ'-repmat(Fpt',1,m);  % get Sn-S*

        AAind = 1:l-1-delay;
        BBind = 1+delay:l-1;

        for ke = 1:l-1-delay
          if exist('skips') && sum(AAind(ke)==skips)
            AAind(ke)=0;
            BBind(ke)=0;
          end

          if exist('skips') && sum(BBind(ke)==skips)
            BBind(ke)=0;
            AAind(ke)=0;
          end
        end



        AAind=deldup(AAind);
        BBind=deldup(BBind);

        AA = RQ(:,AAind);
        BB = RQ(:,BBind);

        J2L = AA*pinv(BB);

        EFM(gc-1,dela) = max(abs(eig(J2L)));
    end


        clear skips

        %pause
        clf
    end  % for each gait cycle

else



end  %if we want to do each gait cycle
```

```
function D = dstptpln(plpoint,N, somepoint)

D=abs(dot(somepoint-plpoint,N))/norm(N);



function D = dstptln(P1, P2, P3)

u = dot( (P3-P1),(P2-P1) ) / sum((P2-P1).^2)  ;

P4 = P1+u*(P2-P1);

D = -(P4-P3);
```

sosbmaxemg.m

```
function [EFM VAR DRMS Poincare] = sosbmaxemg(DATA, G,  LHS, GCC)

    EFM=0;
    AlphaTS=0;
    AlphaRT=0;
    VAR=0;
    EFM=0;
    MPF=0;
    SDT=0;
    devDATA=0;
    DRMS = 0;


    %%calculate DRMS
    [m,n]=size(DATA);
    DATAa = DATA - repmat(mean(DATA,1), m ,1);

    DRMS = (mean(sum(DATAa.^2,2)))^.5;




format compact
%% sosbmaxemg.m
%% calculates stochastic orbital stabilty, instantaneous FM, and
%% Variability in EMG.  Ignores "spike" points
%% MArch 12, 2007, Hyun Gu Kang
%% Last modified:  May 29, 2007
```

```
%% taken in n-dimensional state space matrix
%% inputs:  DATA = matrix of data, each column = state variable, and each
%% row is time point
%%         LHS = Left heel strike data (or other demarkations of cycles
%%         GCC = the gait cycle parts of interest: i.e, 1:100 for all, or
%%         30:40 if you only want to look at a particular % gait cycle.

%%  Dependent Measures

EFM = zeros(100,1);

%downsample
[m,n]=size(DATA);
faze = zeros(m,1);



for S=1:n %for each state variable

  %% Finite fourier series
  xo = mean(DATA(4001:end,S));  %get the center


  % Normalize
  NumStrides = length(LHS)-1;
  Strides=zeros(NumStrides-1,101);

  for i = 1:NumStrides-1
    BeginStrideSamp = LHS(i);
    EndStrideSamp = LHS(i+1);

    if EndStrideSamp>m
      break;
    end
    [NormStride,time] = normalfuLCM(DATA(BeginStrideSamp:EndStrideSamp,S));
    %[FineStride,time] = normalfuLCM(DATA(BeginStrideSamp:EndStrideSamp,S),1e3);
    Strides(i,1:101) = NormStride;
    %FineStrides(i,1:1e3) = FineStride;
    faze(BeginStrideSamp:EndStrideSamp) = linspace(0,100,EndStrideSamp-BeginStrideSamp+1);

  end %-- for i = 1:NumStrides


  Str = reshape(Strides', 1, numel(Strides));
   Amaaa = mean(Strides,1)';%[ xref(99) xref ]; %size = 102 [ 99% 0% .... 99% 100%]

  Amean(:,S)= [Amaaa(95:100,1); Amaaa(:,1); Amaaa(2:7,1)];

end % S
```

```
%figure(1)


% for defining the section, use 99%- 0/100% - 2 % .... 99%, 100%.  Sections
% defined only at 0-99%.  100% assumed to be equal to 0%

%define Poincare section as a line perpendicular to the points, finds slope
%of things crossing the line and plots them as proposed originally
K = [1,2,5,10,20,30,40];  FM = zeros(100,1);
clear S

TS=1;
AlphaTS=0;

if TS==1

  for gc= GCC+1       %gc = 2 really is 0%, so we study 0%-99%
    Current_gc = gc-1

    figure(1)
    %subplot(2,2,1)
    cla
    %plot3(DATA(:,1),DATA(:,2),DATA(:,3),'c','LineWidth',0.5)
    hold on
    %plot3(Amean(:,1),Amean(:,2),Amean(:,3),'r.-','LineWidth',3)
    hold on

    %step 1: determine flow vector, T in the document
    %T = (amean((gc-1)*100+1,:)) -amean((gc-1)*100-1,:);
    T = Amean(gc+1,:)-Amean(gc-1,:);

    % step 2:  define S*n, current fixed point
    Sn = Amean(gc,:);

    % Method 1:  find points right before and right after the Poincare section

    %1. find cluster of points nearby the "fixed point"
    [m,n]=size(DATA);
    %minpt = zeros(7000,101);


    dist=sum((DATA-repmat(Sn,m,1)).^2,2);  %calculate (Euclidian distance)^2 between fixed point and
all points in the data
    Youter = dist<100^2*3;  % 0.05 is adequate for this simulation of this stochastic van der Pol
    sumYo = sum(Youter);
    Yinner = dist<200^2*3;
    %Use phase information instead:
    %A1 = r% before the current GC
    %A2 = r% after the current GC
```

```
r = 5;
A1= gc-1-r-2;
A2= gc-1+r;

if A1 <0
   Y1 = faze>(A1+100);
   Y2 = faze<A2;
   Y = Y1+Y2;

elseif A2 >100
   Y1 = faze>(A1);
   Y2 = faze<(A2-100);
   Y = Y1+Y2;
else

   Y1 = faze>(A1);
   Y2 = faze<(A2);
   Y = Y1.*Y2;
end

Y=Y.*Youter;%+Yinner;
sumY=sum(Y);



%subplot(2,2,2)

% ylim([0.5 1.5])
hold on
%subplot(2,2,1)
a=0;
clear NN
for j = 1:m
   if Y(j) >0
      a=a+1;
      NN(a)=j;
   end
end
clear a
a=0;
clear NN2
for j = 1:m
   if Yinner(j) >0
      a=a+1;
      NN2(a)=j;
   end
end
clear a

NN = [NN-2, NN, NN-1];
%NN=[NN-10, NN-9, NN-8, NN-7, NN-6, NN-5, NN-4, NN-3, NN-2, NN-1, NN, NN+1, NN+2,
NN+3, NN+4, NN+5, NN+6, NN+7, NN+8, NN+9, NN+10, NN+11];
```

```
NN=NN.*(NN>0).*(NN<=m);
NN=sort(NN);
NN=deldup(NN);

NNlength = length(NN);
[M]=length(NN);
%NN = sample#'s of points nearby the fixed point
kay = 5;


Z=zeros(m,1);
%find points in NN2 that are not in NN
a=0;
clear NN3
for o=1:length(NN2)
   if (sum((NN2(o))==NN))==0
      a=a+1;
      NN3(a) =NN2(o);
      Z(NN2(o)) = 1;

   end
end
clear a

minpt_raw=zeros(M,1);

for j = 1:M %for each point in NN
   hold on

   %
   clear D Br cost D2
   D=0; cost=0; D2=0;

   for k = 1:kay
      %figure(2)
      if NN(j)+k-1 > m
         break
      end
      Sk =DATA( NN(j)+k-1,:);
      VPD = Sk - Sn;  %vector from P to data
      cost(k) = dot (T,VPD) /norm(T)/norm(VPD);
      D(k) = dstptpln(Amean(gc,:),T, DATA(NN(j)+k-1,:)); %track distance as it changes while we
follow the trajectory
      D2(k)= norm(Sn-DATA(NN(j)+k-1,:));
   end

   [y,I]=max(cost>0);

   [y,I2] = min(D);
   [y,I3]= min(D2);

   if abs(I-I3) > 4
```

```
        break
    end


    if I>1 && I<(length(cost)) && sum(NN(j)+I-2 == NN )

        minpt_raw(j) = NN(j)+I-1-1;
    end

end %  j


% get rid of duplicates

minpt  =deldup (minpt_raw);
minpt = sort(minpt);

l = length(minpt);

%fill in "missing" minpt and eliminate minpt too close to each other

ADM = round(mean(diff(LHS)));
DM = diff(minpt);

while max(DM)>1.6*ADM
    for v=1:length(minpt)-1
        if DM(v) >1.6*ADM
            minpt=[minpt;minpt(v)+ADM];
        elseif DM(v) <.5*ADM
            minpt(v)=0;
        end
    end
    minpt  =deldup (minpt);
    minpt = sort(minpt);
    DM=diff(minpt);
end

for v=1:length(DM)
    if DM(v) <.8*ADM
        minpt(v)=round(minpt(v)+.2*ADM);
    end
end

for v=1:length(minpt)-1
    if DM(v) <10
        minpt(v+1)=0;
    end
end

if max(minpt) > m
    [wai,ai]=max(minpt);
```

```
    minpt(ai) =0;
end


minpt  =deldup (minpt);
minpt = sort(minpt);
l = length(minpt)

q=0;
%check minpt to see that it is in fact right before the plane
clear cost costp
for j=1:l

    oldpt=minpt(j);

    VPD = DATA( minpt(j),:) - [Amean(gc,:)]; %vector from P to data
    cost(j) = dot (T,VPD) /norm(T)/norm(VPD);

    while cost(j)>0

        minpt(j)=minpt(j) -1;

        %plot3(DATA(minpt(j),1),DATA(minpt(j),2), DATA(minpt(j),3),'m*')



        VPD = DATA( minpt(j),:) - [Amean(gc,:)];  %vector from P to data
        cost(j) = dot (T,VPD) /norm(T)/norm(VPD);
        q=q+1;
    end

    %drawnow


    if (minpt(j) <m)

        VPD = DATA( minpt(j)+1,:) - [Amean(gc,:)]; %vector from P to data
        costp(j) = dot (T,VPD) /norm(T)/norm(VPD);

        while (costp(j) <0) && (minpt(j)<(m-1))
            minpt(j) = minpt(j) +1;

            %plot3(DATA(minpt(j),1),DATA(minpt(j),2), DATA(minpt(j),3),'m*')

            VPD = DATA( minpt(j)+1,:) - [Amean(gc,:)];   %vector from P to data
            costp(j) = dot (T,VPD) /norm(T)/norm(VPD);
            q=q+1;
        end

        %drawnow
```

```
    end




    if abs(minpt(j)-oldpt)>15
        minpt(j)= inf;
    end
    q=0;

  end


  prept = minpt;
  l=length(prept)

  %%now we have all the points right before it crosses the poincare
  %%section

  %%%% modifications specific to EMG, with G vector to indicate
  %%%% spikes

   % list of bad points
  a=0;
  clear BadPT
  for j=1:l
    if G(j)==1
        a=a+1;
        BadPT(a)=j;

    end
  end

% Mask out unusable points with Inf

 a=0;

 if exist('BadPT')

  for j=1:l
    if sum(prept(j)==BadPT)>0
        prept(j) = inf;

    end
  end
 end
```

```
  %% skips
  a=0;
  clear skips
  for j=1:l
     if prept(j)==inf
        a=a+1;
        skips(a)=j;
     end
  end


for j=1:l

   if prept(j) >m-1
      prept(j)=0;
   end
end

prept=deldup(prept);
 l = length(prept);

  preptend=prept(end);

  %% Calculate dependent measures

  clear Ixs x costhet DD Sigma FM

  for j=1:l %for each cycle

     if (prept(j) ~= inf) && prept(j)<18301

        %find intersection of trajectory and Poincare section
        %plane is defined by T dot (x-S*)=0,
        %line is defined as x = P1+u(P2-P1)

        uu = dot(T, Sn-DATA(prept(j),:))/dot(T,DATA(prept(j)+1,:)-DATA(prept(j),:));
        Ix = DATA(prept(j),:)+uu*(DATA(prept(j)+1,:)-DATA(prept(j),:));

        Ixs(j,:) = Ix;    % so Ixs is the state exactly at a % gait cycle

        DD(j,:) = Ix-[Amean(gc,:)]; % D vector in proposal
        xkx(j) = norm (DD(j,:));

     end

  end %j

% store intersections in a STRUCT
```

```
Poincare(gc-1).ix = Ixs;
Poincare(gc-1).FP = Amean(gc,:);

    %variability
    VAR(gc-1) = sqrt(mean((deldup(xkx)).^2));  % RMS distance from fixed point



%Extended Floquet Multiplier

for dela = 1:1

    delay = (dela);  %extended Floquet Multiplier
    %need to account for non-consecutive data:
    RQ = Ixs;

    [m,n]=size(RQ);
    Fpt = Amean(gc,:);   %this is S*
    RQ=RQ'-repmat(Fpt',1,m);  % get Sn-S*

    AAind = 1:l-1-delay;
    BBind = 1+delay:l-1;

    for ke = 1:l-1-delay
        if exist('skips') && sum(AAind(ke)==skips)
            AAind(ke)=0;
            BBind(ke)=0;
        end

        if exist('skips') && sum(BBind(ke)==skips)
            BBind(ke)=0;
            AAind(ke)=0;
        end
    end



    AAind=deldup(AAind);
    BBind=deldup(BBind);

    AA = RQ(:,AAind);
    BB = RQ(:,BBind);

    J2L = AA*pinv(BB);

    EFM(gc-1,dela) = max(abs(eig(J2L)));
end
```

```
        clear skips

        %pause
        clf
    end  % for each gait cycle

else



end  %if we want to do each gait cycle



function D = dstptpln(plpoint,N, somepoint)

D=abs(dot(somepoint-plpoint,N))/norm(N);



function D = dstptln(P1, P2, P3)

u = dot( (P3-P1),(P2-P1) ) / sum((P2-P1).^2)  ;

P4 = P1+u*(P2-P1);

D = -(P4-P3);
```

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Vita

Hyun Gu Kang was born on October 27, 1978 as 강현구(姜峴求) to Paul Hoshin Kang and Bok-Hee Lee Kang, in Seoul, Korea. After growing up in and around London, Seoul, Dhaka, and Chicago, he graduated from New Trier High School in Winnetka, IL in 1996. He received a B.S. in Chemical Engineering with Departmental High Distinction at University of Illinois at Urbana-Champaign in January 2001. He received a M.S. in Biomedical Engineering from University of Virginia in Charlottesville, VA in January 2003, and then entered University of Texas at Austin in August, 2003. He was naturalized as a U.S. citizen in 2001. He is currently a post-doctoral fellow at Hebrew SeniorLife Institute for Aging Research, and a research fellow in medicine at Harvard Medical School in Boston, MA. He would like to pursue a career in biomechanics and geriatrics research and policy. He enjoys performing both early music and 20th century choral music, swing dancing, watching Lost, Heroes and Good Eats, and keeping track of the airline and health care industries.

Permanent address: 1054 Countryside Drive, Walnut CA 91789

This dissertation was typed by the author.